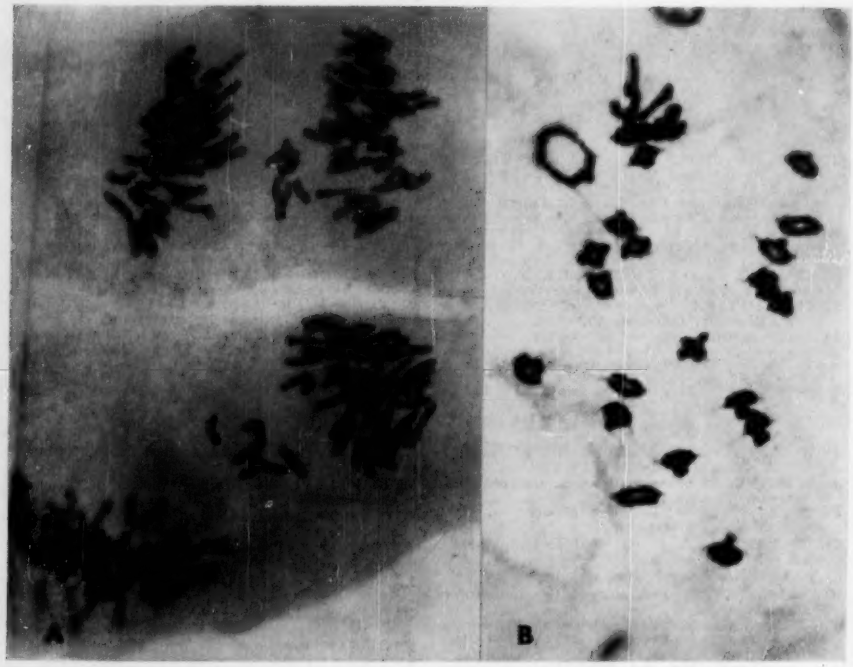
The Journal of HEREDITY



CHROMOSOMES OF A COLCHICINE-INDUCED OCTOPLOID

(SEE PAGE 297)

Cordovan Mice

More Tongue Rollers

Effects of Tomato Gene *bi*

Male Influence on Sex Ratio in Mice

VOLUME 46 NOVEMBER-DECEMBER, 1955 NUMBER 6

The American Genetic Association

An incorporated organization devoted to promoting a knowledge of the laws of heredity and their application to the improvement of plants, animals, and human racial stocks

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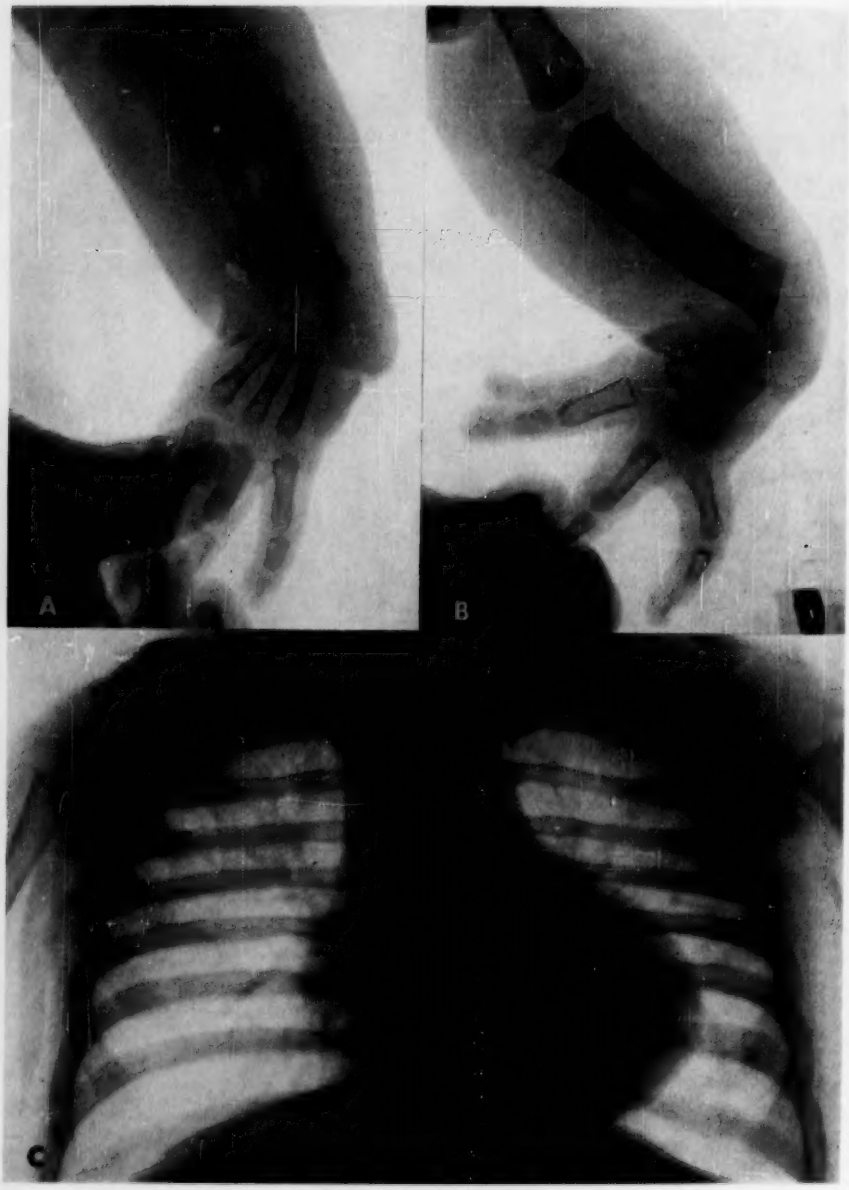


FOREARMS, HANDS AND CHEST OF INDIVIDUAL IV-27

Figure 1

A—The X-ray shows no trace of a radius, two ossification centers in the carpus, absence of first metacarpal, rudimentary second metacarpal, and normally sized and shaped third, fourth and fifth metacarpals. There are only three fingers with three phalanges to each. *B*—There is radio-ulnar synostosis on the left forearm, the proximal and distal ends of the radius and the ulna are entirely fused. Two ossification centers are seen in the left carpus. Only the third, fourth and fifth metacarpal bones are seen on the left hand, but there are four fingers, each with three phalanges, the index being rudimentary and fused with the third finger. *C*—The X-ray of the chest shows the apex of the heart rounded and lifted above the diaphragm.

HEREDITARY MALFORMATIONS OF THE UPPER EXTREMITIES

In Three Generations

EMMANUEL MARGOLIS AND ELIJAH HASSON*

THE exact mode of inheritance in the present family, showing such a wide variation in physical expression of a skeletal trait, is not fully interpreted in this study. Nevertheless we are reporting it, encouraged by Stiles and Dougan,[19] who say: "Our knowledge of human genetics will be most rapidly advanced, not by reporting isolated cases that can be easily analyzed, but rather by reporting all possible types of heredity, regardless of whether they lend themselves to a simple explanation or not."

Since the malformations of the upper extremities in this study show so much variation in expression, it seems necessary to discuss the members of the pedigree individually.

In the present family the first known appearance of abnormality was established in two siblings whose parents and grandparents had no recognizable defects.

The first of the affected sibs, a female, (II-5 of the pedigree), died at the age of 61, unmarried. Little is known about her anomaly but she is reported to have had only four fingers on each hand.

The second sibling, a male (II-6), is now 57 years of age. The deformities to be found on his upper extremities are as follows:

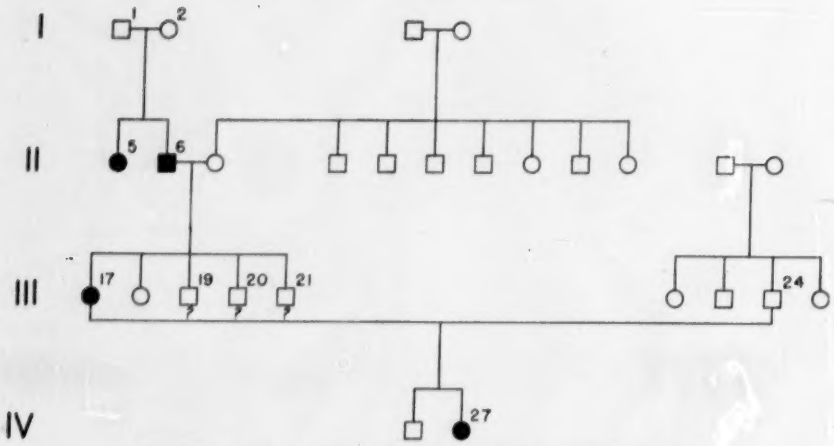
The arm and forearm of the right upper extremity can be easily distinguished; together they are shorter than normal. There are only four fingers on each hand, the thumb is absent, the fourth and especially the fifth finger are bent radially (clinodactyly) and the hand is bent forward. The X-ray film (Figure 3), reveals the forearm with no visible abnormalities, the radius and ulna seem normally developed. There are several carpal bones lacking, and, the thumb is completely absent.

The upper left extremity extends about one-half the way down the chest wall. The hand is bent inward, and the thumb is absent. The index finger is curved, shorter than the other fingers. The fifth finger is sharply bent toward the index. The X-ray (Figure 3), shows total absence of the forearm (hemimelia) and an underdeveloped carpus joined to the distal part of the humerus. The carpal bones seem to be fused with the metacarpal. There is also synostosis among the proximal-medial and medial-distal phalanx of the index (symphalangism). It is permanently bent in the direction of the flexure (camptodactyly).

This member of the pedigree married II-7, who is at the moment a woman aged 53 and found by the authors to be free of skeletal abnormalities. Her parents as well as seven siblings are also free of known malformations. Five children were born of this marriage: the first, III-17, revealed skeletal deformities on both upper extremities, the others are reported to have been born normal, but three of them died in infancy—of pneumonia at the age of one month, of heart disease at nine months and of asphyxia after birth.

The upper extremities of III-17 are shown in Figure 4. The length of the right arm is 20 cm., of the forearm, 10 and of the hand, about 15 cm. The length of the left arm is 18 cm., of the forearm,

*Hadassah University Hospital-Medical Outpatient Dept. "C", Jerusalem and Tel Aviv, respectively. The authors wish to express their sincere appreciation to Dr. S. Schorr and Dr. G. Breslaw, the X-ray Department, Hadassah University Hospital, for reading the X-ray films. Thanks are due to Prof. Th. Grushka, Department of Social Medicine, and Dr. E. Goldschmidt, Department of Zoology, for their helpful suggestions.



A PEDIGREE OF MALFORMED UPPER EXTREMITIES

Figure 2

The deformities appear in three generations; males and females are both affected.

16 cm., of the hand, about 17-18 cm. The thumb is absent on both hands, which are held pronated.

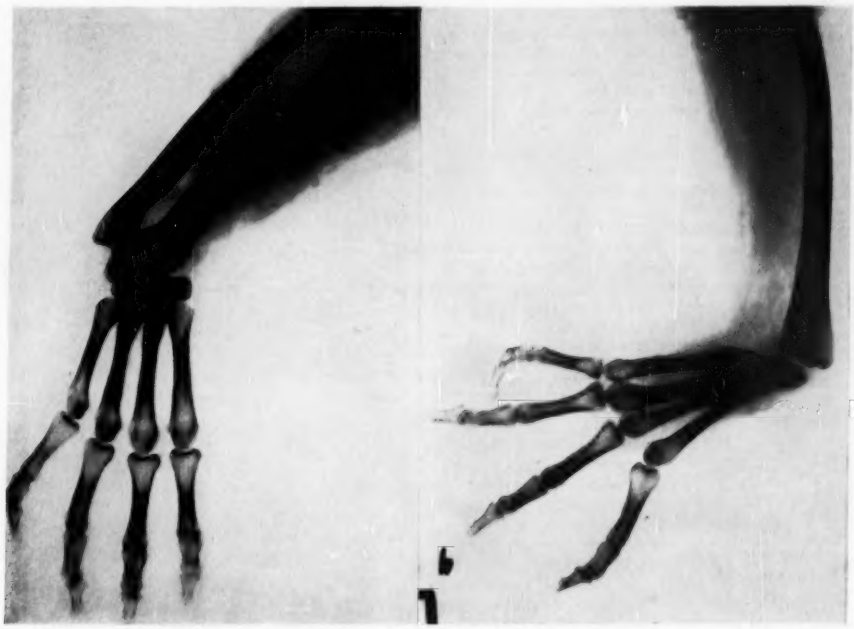
The X-ray film of the right forearm (Figure 5) reveals total aplasia of the radius; the fossa olecrani is flattened, the shaft of the ulna is broadened, shortened and curved. The distal part of the ulna is flattened too, no styloid process is seen. The hand articulates with the ulna by means of a false joint on its outer border. There is fusion of the upper and lower row of the carpal bones. Partial fusion of the soft tissue (without fusion of the bones), and not extending the entire length of the digits, is to be noted; hence the fingers of the right hand seem shorter than those of the left hand. There is no visualization of thumb metacarpal and phalanges and the fifth finger is radially curved.

The X-ray film (Figure 6), shows a normal distal end of the left humerus; the articulating surfaces of the distal part of the radius and ulna are flattened and abnormal in shape. There is fusion of several carpal bones and some carpal bones (scaphoid and trapezodail) seem to be absent. No traces of the thumb metacarpal and the phalangeal bones can be seen. The length of the metacarpus together equals the length of the forearm. The fifth finger is radially curved.

The marriage of III-17 to the normal III-24 (whose parents and siblings are stated to be normal) produced two daughters. The first of these, now four years old, exhibits no deviation from the normal. The second is now two years old. Her upper extremities are shown in Figure 7. The right arm, forearm and hand measure 9, 6, and 8 cm. respectively. The left arm, forearm and hand are 12, 7, and 8 cm. long, respectively. There are only three fingers on each hand. They correspond probably to the third, fourth and fifth finger. A rudimentary index, closely fused with the third finger, can be distinguished on the left hand. The finger is mesially curved.

The X-ray of the right side (Figure 1*A*), shows no trace of a radius, two ossification centers in the carpus, absence of the first metacarpal, a rudimentary second metacarpal and normally sized and shaped third, fourth and fifth metacarpals. There are only three fingers with three phalanges to each metacarpal.

The X-ray (Figure 1*B*), reveals radioulnar synostosis on the left forearm, the proximal and distal ends of the radius



RIGHT AND LEFT FOREARM AND HAND OF INDIVIDUAL II-6

Figure 3

At the left is shown the right forearm of II-6 with no visible abnormalities. The radius and ulna seem normally developed. There is lack of several carpal bones and the thumb is completely absent. At the right is shown the left forearm and hand of the same individual. Total absence of the forearm and an underdeveloped carpus joined to the distal part of the humerus can be seen. The carpal bones are fused with the metacarpal, there is also synostosis among the proximal-medial and medial-distal phalanx of the index finger (symphalangism)—it is permanently bent in flexure (camptodactyly).

and the ulna being entirely fused. Two ossification centers are seen in the left carpus. Only the third, fourth and fifth metacarpal bones are seen on the left hand, but there are four fingers, each with three phalanges, the index being rudimentary and fused with the third finger.

The X-ray of the chest (Figure 1*C*), shows the apex of the heart rounded and lifted above the diaphragm. No clinical evidence of heart disease was found.

Discussion

Many congenital abnormalities of the upper extremities are found in the literature, as stated by Stiles and Dougan.[10] Most of them especially affect the hand, in which, according to Gates,[11] more abnormalities are known than in any other part of the body, with the exception of the eye. Kemp[13] also mentions the fact that malformations of the arm are rarer than malformations of the hand.

Barsky,[2] based on a survey of the medical literature of the last 30 years, confesses that he has been unable to find any valid statistics of the occurrence of congenital hand anomalies. Birch-Jensen[3, 4] in Denmark found one in 22,000 children born with congenital absence of the forearm and one in 6,438 persons with hand anomalies (excluding polydactyly and syndactyly). In the Hadassah University Hospital in Jerusalem during the last 15 years 12 cases were recorded as follows: polydactyly—5 cases; syndactyly—3; finger deformities—2; radio-ulnar synostosis—1; and clubbed hand—1 case.

It is not uncommon that hereditary skeletal anomalies exhibit considerable variation, and there is also a wide degree of variation in the extent to which the persons in our pedigree were deformed: The grandfather has his left forearm totally absent, the daughter exhibits

absence of right radius and so does the granddaughter, but there is also radio-ulnar synostosis in her left forearm. It is difficult even to decide into which main category these three cases belong. The only common trait found in both hands of all three affected members of the pedigree is the oligodactyly and clinodactyly; but they are combined with a number of other, related or unrelated limb malformations, which differ in each member of the pedigree.

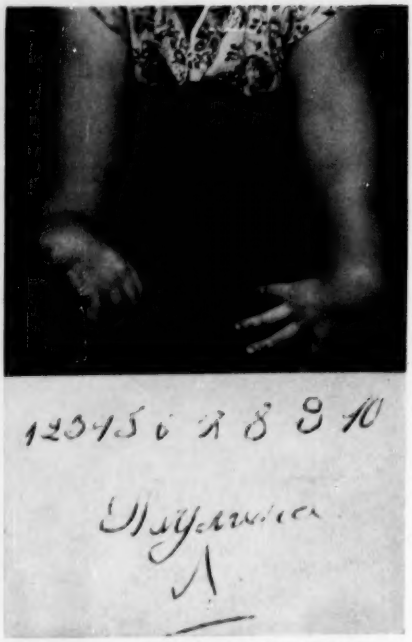
Oligodactyly seems not to be very frequent. Gates[11] mentions some cases: one family, cited by Veit,[23] another—showing oligodactyly for five generations, cited by Schroeder[17] and a third one—the pedigree reported by Stiles and Dougan.[19] In his series of 62 Barsky[2] had seven cases of complete absence of a finger, 3 bilateral and 4 unilateral. According to Kemp[14] this anomaly "occurs to all appearance sporadically, but in some families it shows dominant inheritance."

Oligodactyly may also be seen together with syndactyly—"the most common congenital deformity of the hand" (Barsky[2]), but this latter condition is more often associated with polydactyly, as stated by Barsky[2] and Gates.[11]

According to Barsky,[2] syndactyly frequently accompanies not only poly- or oligodactyly but also deficiencies of the long bones. In his series of 62 cases, 29 had syndactyly, 11 of them combined with other congenital anomalies of the upper extremity. According to him syndactyly in humans is most frequent between the middle and ring finger and almost twice as frequent in males as in females. Davis and German[10] also state that the literature on syndactyly shows a predominance of males, and in their own particular series of 50, the males made up 68 percent of the total.

Syndactyly may involve more than two fingers, sometimes all of the fingers. The condition not only varies in the number of digits involved but also in degree. Thus syndactyly may be extending to the tip of the finger—first degree; extending half way—second degree; barely perceptible at the base—third degree. Sometimes the bones are fused (synostosis) and even the tendons and nerves are joined, but the most common type is simple skin webbing. Such was the case with III-17 of the present pedigree (Figures 4 and 5). In IV-27 of the present pedigree the syndactyly involving the index and middle fingers of the left hand, was of second degree, the bones were not fused and one of the webbed fingers (the index) was rudimentary (Figures 7 and 1*B*).

Symphalangy—fusion of the interphalangeal joints, usually of the middle phalanx with the proximal and distal—occasionally accompanies syndactyly.[2] However we have not found it in the members of the pedigree which exhibited syndactyly. But we did see it in both hands of II-6, who did not exhibit syndactyly at all.



THE MALFORMATION AND HANDWRITING OF III-17

Figure 4

The upper photograph shows the shortened forearms. The thumb is absent in both hands, which are held pronated. In the lower photograph is a sample of the same individual's handwriting.

Clinodactyly—fingers bent in the radial direction, "crooked little fingers"—were found in every one of the affected members of the pedigree. Many cases of clinodactyly are cited by Gates.[11] We also have more than once traced crooked little fingers, non-combined with other anomalies, through three generations.

Every one of the affected members in the present pedigree revealed defects in one or both forearms. In the first of them, II-6, there was total absence of the radius and ulna of the left side. According to Birch-Jensen[6] this is a rare deformity, observed in Denmark in only one of 22,000 children, and only unilaterally. He reports a unique case of bilateral partial absence (distal part absent) of both antebrachial bones and another one of bilateral absence of the radius.

There is not a single case of total absence of both forearm bones in any of the other affected members of the present pedigree. III-17 possesses a normal left forearm. The radius is absent in her right forearm. The same de-

RIGHT FOREARM AND HAND OF III-17
Figure 5

The radius is absent, fossa olecrani is flattened and curved. The distal part of the ulna is also flattened, no styloid process is seen. The hand articulates with the ulna by means of a false joint on its outer border, there is fusion of the upper and lower row of the carpal bones. Partial fusion of the soft tissue (without fusion of bones), not extending the entire length of the digits, is to be noted; hence the fingers of the right hand seem shorter than those of the left hand. The thumb is entirely absent and the fifth finger is radially curved.

fect is found in the same place in her second daughter—IV-27. In general, total or partial absence of the long bones are rare defects (Kemp[14]). Among them, however, the congenital absence of the radius in part or whole is, according to Barsky,[2] fairly common. He found it once in his series of 62. It was not associated with oligo- or polydactyly. Kato[13] collected 253 cases in the literature up to 1923, including three cases of his own. Birch-Jensen,[3] as mentioned above, reports a case with bilateral absence of the radius. This is an extremely peculiar case—carpal bones are missing and there is aplasia of digits (only one digit is present). Both cases of the missing radius in our pedigree were associated, as usual, with shortened, thickened and curved ulna, radially deviated hand, almost at a right angle to the long axis of the forearm.

IV-27 of the pedigree exhibits another peculiar deformity, namely synostosis of the radius and ulna in the left forearm. Radio-ulnar synostosis is not very rare, according to Gates.[11] Stiles and Dougan[19] also state that cases of congenital radio-ulnar synostosis have been frequently reported. This is somewhat in contradiction with Cohn[8] who points out that "radio-ulnar synostosis is a rare developmental anomaly." Davenport *et al.*[9] made an extensive study of this condition. It is found to be bilateral in about half the cases and more common in males. In the pedigree of Davenport *et al.*,[9] 12 of the 13 affected cases were males, which might suggest that the defect may be in the Y-chromosome with an occasional crossing-over to the X. Cohn[8] also found radio-ulnar synostosis more common in males. In some other pedigrees there was an excess of affected males too, and the inheritance was irregularly dominant, while in the pedigree reported by Davenport and his colleagues[9] it showed clear dominance. We, however, found it in only one of the three affected members and this was a female (IV-27).

While confirming Kemp's[14] opinion that congenital deformities of the forearms tend to appear together with other malformations in the carpal and digital bones, our study does not seem to be in accordance with another of Kemp's statements that very often these deformities are associated with other anomalies, namely—heart, spinal and thoracic deformities and/or mental deficiency. The members of the present pedigree did not reveal any of these conditions. It is true, however, that we lack accurate data concerning siblings III-19, -20 and -21; and it is quite possible that they were affected by deformities of this sort which were even responsible for their premature death.

The etiologic factors in congenital anomalies of the upper extremities have not been conclusively established. However the existence of a definite hereditary tendency has been reported by many authors. Barsky,[2] in his own series, found indications of hereditary factors in five percent of the cases. In 1916 Boorstein[5] expressed the opinion that bilateral congenital malformations can generally be considered to be of a genetic nature while unilateral congenital malformations are probably due to prenatal trauma. This definite statement was not confirmed by other authors. Brown,[6] while reporting an isolated case of malformed upper extremities and thorax, agrees that "it seems likely that any such bilaterally symmetrical deformity (as reported by Stiles and Dougan[19]) would be the result of a widely generalized developmental mechanism such as might be expected to be present if the condition were hereditary." Bagg[1] states: "Abnormalities of the limbs are definitely inherited."

It is not uncommon for hereditary malformations of the upper extremities to exhibit considerable variation, including bilateral dif-

LEFT FOREARM AND HAND OF III-17
Figure 6

The articulating surfaces of the distal part of the radius and ulna are flattened and abnormal in shape. There is fusion of several carpal bones. No traces of first metacarpal and phalangeal bones can be seen. The fifth finger is radially curved.

DEFORMED UPPER EXTREMITIES OF IV-27
Figure 7

The forearms are shortened and curved. There are only three fingers on each hand. A rudimentary index, closely fused with the third finger, can be distinguished on the left hand. The fifth finger is mesially curved.

ferences in expressivity, as shown by Stiles and Weber,[21] and Stiles[18] and Stiles and Pickard.[20]. Bunnel[7] also has seen "that certain types of deformity may be transmitted in pure form with only slight variations through many generations while in other families there is a predisposition to deformity in a wide range of types." This situation makes it difficult to establish genetic factors, and the cause of the variability of the phenotypes thus produced remains unknown. As shown, a variation in the deformities of the persons in the present pedigree was found to such an extent that it might be reasonable to ask, as Brown[6] does, if one is justified in treating the data as if there were only two classes—normal and malformed.

The following facts of genetic interest can be established in the present case:

The deformity is stated to be absent in generation I of the present pedigree. After its first (?) appearance in generation II it behaves like a dominant trait with variable expressivity and without any evidence of sex linkage. In view of the lack of information regarding the generation preceding II we are unable to state whether we are dealing with a new mutation having occurred in the sex-cells of either I-1 or I-2, or with a hereditary trait exhibiting incomplete penetrance. Limb-malformations which are inherited as dominant with incomplete penetrance are on record (Stiles and Dougan,[19] Stiles and Pickard[20]), although none of the pedigrees cited in the literature includes the particular assortment of skeletal abnormalities that characterize the present family.

Whatever the exact cause of the peculiar anomalous development in the present case, it seems reasonable to suppose that it began to operate at least as early as the fifth week after conception. According to Lewis,[15] as

early as the fifth week of normal ontogeny the mesenchymal and cartilagineous rudiments of the humerus, radius, ulna, individual carpal bones and elements of the five fingers are recognizable. We are here concerned with the hypothesis proposed by Stockard,[22] confirmed by Bagg[1] and strongly supported by Gruenfelder,[12] namely that the time when the causa efficiens occurs during pregnancy is of much greater importance for the development of congenital anomalies than the kind of acting factors.

We deem it of interest to mention some psycho-sociological aspects of this case.

The three affected members of the pedigree do not seem to be socially introverted personalities. Neither of the two adult members of this family avoided either marriage or parenthood. It is not due to the fact that they did not really understand that the defect was hereditary. At least for III-17 it was established beyond any doubt that she did realize the situation and was deeply concerned during her first and second pregnancy with the possibility of bearing anomalous children. The birth of the affected child was a serious disappointment to her, but she very soon recovered and is now a devoted mother to her normal as well as to her maldeveloped daughter.

The grandfather, II-6, was relatively the most un-cooperative patient. He refused, for example, to have his upper extremities photographed. The authors had the impression that he deliberately concealed details of his family history; and it might be that in the three children who died in early infancy there were some anomalies.

The little child, IV-27, appears not only healthy and normal but even attractive, except in respect to the defect in her upper extremities. In none of the three affected persons was the defect correlated with low mentality or psychopathy.

We were able to confirm the statement made by Birch-Jensen[3] namely that this kind of patient presents no special social problem as they are usually intelligent and undergo training with considerable zeal, even for occupations for which we should not expect them to be well suited. The grandfather in this family, for instance, runs a grocery, the daughter looks after her own children and occupies herself with various kinds of handicrafts and is even able to write, as shown in Figure 4. It is interesting to see how the little granddaughter, attracted by toys, is successfully trying to take and handle them with her maldeveloped limbs.

We feel the necessity of adding a few words on the eugenic problem brought forth by this case: Are we permitted to go so far as to advise against marriage of persons with this kind of malformation or if married, to propose abortion or even sterilization? It is our opinion that in a free society a woman of the mental capacity of III-17 should be left to decide for herself, whether she wishes to give birth to other children after we have explained to her that her chance of having another malformed child is probably close to 50 percent.

And finally some notes concerning the therapeutic prospects of this particular case. Of the three affected members of the family the only one suitable for surgical intervention is the child, IV-27, because of her age. Only the correction of her right forearm (absence of radius) can be considered. In regard to the radio-ulnar synostosis in the left forearm, many operative procedures have been given a trial but all these have failed to produce any real rotation, (Bunnell,[7] Platt[16]).

Literature Cited

1. Bagg, H. J. A morphological study with special reference to the etiology of club-feet, syndactylism, hypodactylism and congenital amputation in the descendants of X-rayed mice. *Am. Jour. Anat.* 43:167-219. 1929.

2. Barksy, A. J. Congenital anomalies of the hand. *Jour. Bone and Joint Surg.* 33-A: 35-60. 1951.

3. Birch-Jensen, A. Two cases of congenital absence of forearm and aplasia of radius. *Ann. Eug.* 17:2-3. 1952.

4. ————. Congenital Deformities of the Upper Extremities. Ejnar Munksgaard, Copenhagen. 1949.

5. Boorstein. *Ann. Surg.* 236-237. 1916; cited in Stiles and Dougan.[19]

6. Brown, E. An isolated human case of malformed upper extremities and thorax. *Jour. Hered.* 34:284-288. 1943.

7. Bunnel, S. Surgery of the Hand. p. 801. J. B. Lippincott, Philadelphia. 1948.

8. Cohn, B. N. E. Congenital bilateral radio-ulnar synostosis. *Jour. Bone and Joint Surg.* 14:404-405. 1932.

9. Davenport, C. B., H. L. Taylor and L. A. Nelson. Radio-ulnar synostosis. *Arch. Surgery* 8:705-762. 1924.

10. Davis, J. S. and W. J. German. Syndactylism (coherence of fingers or toes). *Arch. Surg.* 21:32-75. 1930.

11. Gates, R. R. Human Genetics, Vol. 1, pp. 385-457. The Macmillan Co., New York. 1952.

12. Gruenfelder, B. and W. Lasch. Der Zeitpunkt einer embryonalen Entwicklungsstoerung als ursaechliches Moment kongenitaler Anomalien. *Annales Paediatrici* 173:388-404. 1949.

13. Kato, K. Congenital absence of the radius. *Jour. Bone and Joint Surg.* 6:589-626. 1924.

14. Kemp, T. Genetics and Disease, pp. 203-208. Ejnar Munksgaard, Copenhagen. 1951.

15. Lewis, W. H. Gray's Anatomy of the Human Body, 24 ed., p. 65, Lea & Febiger, Philadelphia. 1942.

16. Platt, H. Modern Trends in Ortho-

pedics, p. 133. Butterworths & Co. London. 1950.

17. SCHROEDER, E. Entshehung und Vererbung von Missbildungen an der Hand eines hypodactyliestaumbaumes. *Monatschr. Geburstth. u. Gynaekol.* 48:210-222. 1918. Cited in Gates.[11]

18. STILES, K. A. The inheritance of brachymetapody. *Jour. Hered.* 30:87-91. 1939.

19. ———— and P. A. DOUGAN. Pedigree of malformed upper extremities. *Jour. Hered.* 31:65-72. 1940.

20. ———— and J. PICKARD. Hereditary malformations of the hands and feet. *Jour. Hered.* 34:341-344. 1943.

21. ———— and R. A. WEBER. A pedigree of symphalangism. *Jour. Hered.* 29:199-202. 1938.

22. STOCKARD, C. R. Developmental rate and structural expression. *Amer. Jour. Anat.* 28: 115-227. 1921.

23. VEIT, G. Uber familiaeres Vorkommen von Oligodactylie. *Zeits. f. Konst-Lehre.* 23: 620-635. 1939. Cited in Gates.[11]

DIFFERENCES BETWEEN FRATERNAL TWINS

IN a recent book review* a statement appeared to the effect that differences between dizygotic twins depended not upon differences between their parents, but upon differences between their grandparents. This is presumably correct for the F_2 from racial crosses, but in any more general application the statement would have to be revised. This illustrates one danger often inherent in observations made on pure lines.

Genetic variation between dizygotic twins (or among siblings) implies heterozygosity in one or both parents. In a population where mating is nearly random, only a minority of the heterozygosity is derived from matings between two homozygous types. Therefore, when a phenotypic difference between DZ twins is due to a single factor, there will often be no corresponding phenotypic difference within either grandparental pair. Furthermore, whenever the twin difference is matched by a difference *between* the grandparental pairs, it will be matched by a similar difference between the parents.

Actually, the parental differences are expected to be more frequent than the grandparental differences. With respect to any one factor in a random-mating population, only a minority of genotypically diverse sibships are derived from matings between two heterozygotes. With respect to incompletely dominant factors, therefore, dissimilar fraternal twins will usually have dissimilar parents. For dominant or recessive traits a more careful statement is necessary. All twin pairs differing in such a trait will have at least one heterozygous (dominant) parent. The other parent, who must carry one recessive gene, will be of recessive or dominant phenotype in proportion to the frequency of the respective genes, except as one phenotype carries a selective disadvantage. Results of the matings are as follows:

MATINGS THAT CAN PRODUCE DISSIMILAR OFFSPRING

Mating Genotype	Mating Phenotype	Relative Frequency	Dissimilar Pairs of Offspring	Relative Frequency
Dd × *dd*	dissimilar	q	*Dd* — *dd*	1/2 q
Dd × *Dd*	similar	p	*Dd* — *dd*	1/4 p
			DD — *dd*	1/8 p

One-half of the fraternal twin pairs from the first type of mating and three-eighths of those from the second type will be phenotypically dissimilar. Hence, if the recessive gene is more than three-fourths as frequent as the dominant gene, i.e., if $q > 3/7$ and $p < 4/7$, most dissimilar twin pairs will have dissimilar parents.

For all traits taken together, the proportion of dissimilar human twins who have dissimilar parents should depend upon the ratio of incompletely to completely dominant traits and on the average gene frequency of the latter. However, for the uncommon recessive, in which parents are likely to be similar, the grandparents are even more likely to be similar. Such parents are all heterozygous, and derived from the following matings:

MATINGS THAT CAN PRODUCE HETEROZYGOTES

Mating Genotype	Mating Phenotype	Relative Frequency	Porportion of Heterozygous Offspring	Relative Frequency
DD × *dd*	dissimilar	pq	1	pq
Dd × *dd*	dissimilar	$2q^2$	1/2	q^2
DD × *Dd*	similar	$2p^2$	1/2	p^2
Dd × *Dd*	similar	2pq	1/2	pq

When only one-half of the dissimilar twin pairs come from similar parents, i.e., when $q = 3/7$, the proportion of these similar parents who came from similar grandparents is $p^2 + pq = p = 4/7$. For rarer recessives, the proportion of similar parents rises, but the proportion of these derived from similar grandparents also rises. The two quantities,

$$\frac{3p}{4q + 3p} \text{ and } p,$$

bear the ratio of $3:(4-p)$.

It therefore appears that in a random-mating human population, differences between fraternal twins should be more highly correlated with parental than with grandparental differences.

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*C. D. Darlington. *Heredity* 8: 288. 1954.

BROWN FEMALE—A SEX-LINKED AND SEX-LIMITED CHARACTER

In the Spruce Budworm

G. STEHR*

THE purpose of this paper is to show that an important part of the intra-specific variability in eastern populations of the spruce budworm, *Choristoneura fumiferana* (Clem.), as this species is delimited taxonomically today[2], is caused by a single gene difference.

While the majority of female moths in a field sample are found to be of various shades of gray, about 15 percent are brown in over-all coloration (Figure 8). Freeman's color plate[2] shows the two types of females as distinctly gray (his Figures 11-13) and ochreous-brown (his Figures 14-16). The difference refers only to the coloration of the upper layer of larger scales (cover scales) on the fore wings, which alone are responsible for the wing pattern, and the scales on the thorax, legs, and the head and its appendages. There is a lower layer of small scales (under scales), which are the only scales on the surface of the hind wings and which become visible on the fore wings, the head, and the thorax only after removal of the cover scales. The color of these under scales is not appreciably different in the two types of females. As it has been shown in the flour moth that cover scales are differentiated from under scales by one or two endomitotic divisions of their cell nuclei[4], the differentiation of gray and brown females is likely to be an effect produced only during the last phase of scale-cell development.

In males no corresponding brown type exists. They are typically gray. However, brown scales can be found on the wings of many males, usually few and widely scattered, but sometimes clustered along veins or grouped in larger numbers in the dark elements of the pattern background, especially the basal, central, and distal fields. Figure 10 of Freeman's color plate illustrates such a male. Similar types also occur among females. Yet, in these cases there are no brown scales intruding into the white and silver-gray areas of the light transversal bands that separate the dark fields, whereas in the females considered as "brown" in this study, these light pattern elements are ochreous, of a lighter, more yellowish shade. Because of this distinguishing feature the occurrence of brown scales, even in considerable numbers, in males and in some females, which here have been called "gray," did not, in our experience, pose any problem in classification; the "brown-female" type is distinct and not connected by intergradations to any other brown color or pattern variants.

A similar color dimorphism, making a portion of the females paler than the rest and having no appreciable effects in the males, occurs in a number of lepidopterous species and has been explained, where analyzed genetically, as being determined by autosomal dominants, limited in expression to the female sex. However, in some cases difficulties arose in obtaining satisfactory examples for the expected 3:1 ratios from matings between heterozygotes, and lethal factors closely linked to the dominant genes had to be invoked to account for the deficiency of homozygous dominants; in turn, it became necessary to assume that rather frequent crossing-over freed the dominant alleles from their associated lethals in those breeding experiments where viable homozygous dominants were obtained. Such a sequence of assumptions appears in most cases to offer the only explanation for the observed

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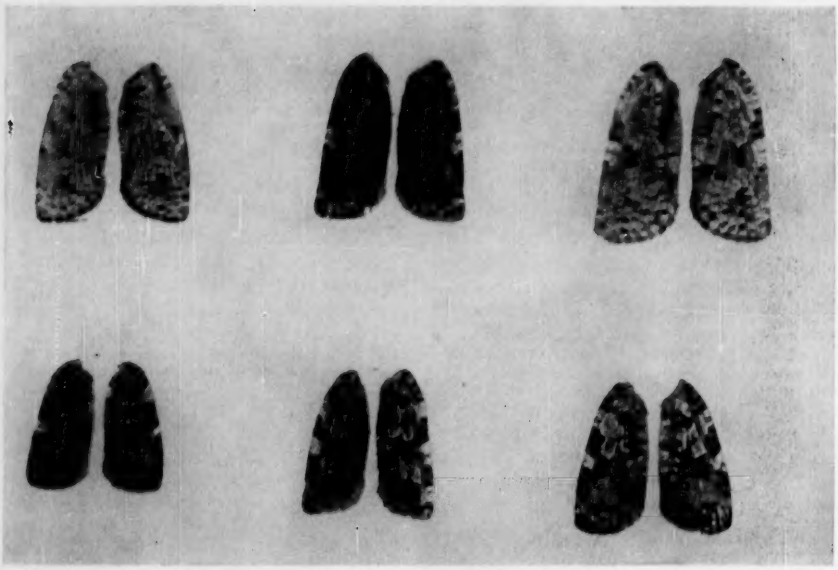

FORE WINGS OF THE SPRUCE BUDWORM

Figure 8

Top row: center, shows a normal gray female. Right and left: brown females. Bottom row: males, brothers of each female, shown above. (Photo by D. C. Anderson.)

facts (see reviews by Ford[1] and Remington[5], with extensive references). However, less complex mechanisms of genetic control of female dimorphism in Lepidoptera are possible, such as sex-linkage with sex-limited gene expression, although this type has apparently not been reported. This is obviously because there are many more autosomal genes than sex-linked ones. Otherwise, there is no *a priori* reason why an autosomal gene should be more readily sex-limited than a gene on the X chromosome; on the contrary, for such groups as Lepidoptera where little or no hormonal control over sex-associated characters exists, a location on the X chromosome seems a rather likely means of restricting gene expression to the heterogametic sex, the preferred sex for the phenotypic exhibition of sex-limited genes.

Breeding Results

In extensive rearings, the technique of which has been described previously[6], inbred lines* soon produced families with all females either gray or brown, so that a simple genetic mechanism could be assumed. The character in question is sex-limited, *i.e.* restricted to the heterogametic females, which obtain a single X chromosome from their father only. Considering the possibility of sex-linkage, the following expectations can be made. If two alleles are available for a locus on the X chromosome, two types of females will be produced. In males three genotypes will then exist, the two homozygotes and the heterozygote. From crosses of homozygous parents, the F_1 females will correspond to the genotype of their father, while F_1 males will be heterozygous. F_2 females will be segregated in a 1:1 ratio and F_2 males will be one-half heterozygous and one-half homozygous for the allele that had been carried by their mother. In half of the F_2 families, then, the females will be of two phenotypes and in the other half they will be uniform, according to the genotype of their F_2 father.

*Initial material kindly donated by Dr. S. G. Smith.

Starting with crosses between purebred families the data of Table I have been obtained. There can be no doubt that the brown female character is determined by a sex-linked and sex-limited gene, *i.e.* it is borne on the X chromosome and expresses itself in the heterogametic females only. Due to this no information about dominance has been obtained, as the relationship between the alleles remains obscured in the male sex, where the gene effects are unknown. This is the first Mendelian gene demonstrated in *C. fumiferana;* the symbol *bf* ("brown female") is proposed for it.

Discussion and Conclusions

The role of the *bf* gene in the dynamics of natural spruce budworm populations is not known. In laboratory rearings, no apparent differences in fecundity, fertility, rate of development, or survival have been found between the two types of females. Biological effects of the gene in its three allelic combinations in males are not as readily assessed but may prove to be of greater interest. The maintenance of dimorphism in natural populations is likely to depend on the balance between selection values for the homozygotes and the heterozygotes, which in this case means that selection among males might determine the relative frequencies of the two phases of females. The frequency of the *bf* gene in Canadian spruce budworm populations east of the prairies may be seen in Table II, which gives the percentages of brown females in mass collections from areas arranged in geographic order from east to west. The *bf* gene appears to be well established at a uniform equilibrium frequency in the whole range of its distribution and hence has probably existed in the field for a considerable time.

The particular color dimorphism of the eastern field populations, which is here established as being due to the *bf* gene, has been recognized by earlier workers, notably A. Gibson[3], who described the moth as having ". . . distinct color varieties . . ." and shows in their correct coloration, the gray and brown types of females in his Figures 2 and 3. He does not mention the sex-limitation of the character specifically, although he implies it indirectly by depicting females only. Freeman[2] seems not to be aware that the ochreous brown female type in eastern spruce budworm populations can be readily distinguished from other brown or brownish variants; he therefore considers merely ". . . a partially sex-associated character with an east-west clinal frequency. . . ." Obviously he is discussing the color polymorphism of *C. fumiferana* as a single and uniform phenomenon throughout the various distribution ranges of the species, including even the much brighter orange-brown and the dark red-brown types that are frequently found in the western group of *fumiferana* populations (west of the prairies and in the Rocky Mountains). As Table II shows, there is no indication of a frequency change along an east-west cline for the *bf* gene.

It appears that the intricate variability of *C. fumiferana* needs to be carefully separated into its morphological and geographical entities, which then have to be analyzed genetically before the complexity and significance of the polymorphism in this species can be fully evaluated.

Literature Cited

1. Ford, E. B. The Genetics of Polymorphism in the Lepidoptera. Advances in Genetics 5: 43. 1953.

2. Freeman, T. N. The spruce budworm, *Choristoneura fumiferana* (Clem.) and an allied new species on pine (Lepidoptera: Tortricidae). *Canad. Ent.* 85: 121-127. 1953.

3. Gibson, A. *In* Swaine, J. M. and F. C. Craighead. Studies on the spruce budworm (*Cacoecia fumiferana* Clem.) Part I. p. 16-17. *Canad. Dep. Agric. Tech. Bulletin* 37, New Ser. 1924.

4. Henke, K. Ueber die verschiedenen Zellteilungsvorgaenge in der Entwicklung des bechuppten Fluegelepithels der Mehlmotte *Ephestia kuehniella. Z. Biol. Zentralbl.* 65: 120. 1946.

TABLE II. Frequency of the *bf* phenotype* in Canadian spruce budworm populations east of the prairies: summer 1953

Regions	No. of Collections	Adult females brown	gray	% brown
Maritime Provinces (N.S., N.B.)	3	35	174	16.75
Quebec	5	18	118	13.24
Northwestren Ontario	7	82	471	14.83
Manitoba, west of Lake Winnipeg	2	33	177	15.71
Total	17	168	940	15.16

*With ♀♀ heterogametic and the *bf* gene sex-linked, the frequency of brown ♀♀ is equal to the gene frequency, provided the gene frequencies in both sexes are equal.

TABLE I. Results of crosses between "brown female" (*bf*) and normal gray (+) stocks in three generations

P		F_1			F_2				F_3		
♀	♂	No. of fam's.	♀ — offspring brown	gray	Genotype of ♂ parents	No. of fam's.	♀ — offspring brown	gray	Genotype of ♂ parents	No. of fam's.	♀ — offspring brown	gray
bf ×	+/+	6	0	76	*bf*/+	14	41	44	+/+	8	0	128
									bf/+	5	50	50
+ ×	*bf*/*bf*	6	39	0	+/*bf*	19	118	123	+/*bf*	14	81	70
									bf/*bf*	7	126	0

5. REMINGTON, C. L. The Genetics of *Colias* (Lepidoptera). Advances in Genetics 6: 403. 1954.

6. STEHR, G. A laboratory method for rearing the spruce budworm, *Choristoneura fumiferana* (Clem.). *Canad. Ent.* 86: 423-428. 1954.

◆

VIABILITY OF DOMINANT WHITE IN THE RABBIT

ROY ROBINSON*

A SEMI-DOMINANT form of white spotting exists in the rabbit. The heterozygote *Enen* is irregularly marked with white areas usually confined to the vental and lateral surfaces of the body. The homozygote *EnEn* is almost entirely white with a few pigmented spots restricted to the head and hind regions of the animal. The eyes are normally fully colored at all times.

Recently, the suggestion has been made that the homozygote may be slightly less viable compared with the normal,[1] Dr. H. Nachtsheim[2] has also commented upon the same feature, contrasting the rabbit gene with the *W* ("black-eyed white") of the mouse.[3]

The sudden demise of Mr. E. C. Richardson prevented a full treatment of his data except for a popular note.[4] The animals in his care were all apparently fed and housed identically at the moment of death (for any cause) of any individual adult.

The chi-square analysis suggests the presence of considerable heterogeneity and reveals that this is concentrated in the excess deaths of *EnEn* homozygotes. A *primae facie* case for lowered viability of the genotype would appear to be evident.

Recorded data on the segregation of the homozygote is sparse; only one group dating from 1919 occurs in the literature (viz. 400 *EnEn*, 827 *Enen* and 415 *enen*).[5] Here the classification is presumably made at an early age and shows no significant departures from expectation due to pre-natal or immediate post-natal mortality ($\chi^2_3 = .362$, $P = .9 - .8$). Information upon the *Enen-enen* comparison abounds and a collation from ten papers of 6,056 animals (3,010 *Enen* and 3,046 *enen*) from backcrosses exhibits no discrepancies ($\chi^2_1 = .214$, $P = .7 - .5$; heterogeneity $\chi^2_9 = 11.105$, $P = .3 - .2$).

Literature Cited

1. RICHARDSON, E. C. Inheritance of white in the English rabbit. *Heredity* 7:150. 1953.

2. NACHTSHEIM, H. Ergebnisse und Problems der Vergleichenden und Experimentellen Erbpathologie. *Jena Z. Med. Naturw.* 76:81-108. 1943.

3. GRUNEBERG, H. The Genetics of the Mouse. 1st Ed. Cambridge University Press. London, p. 62. 1943.

4. RICHARDSON, E. C. *Fur and Feather.* 25th Dec. 1952.

5. CASTLE, W. E. Studies of heredity in rabbits, rats and mice. *Car. Inst. Wash. Pub.* 288:4-28. 1919.

TABLE I. Number of deaths

Genotype	*enen*	*Enen*	*EnEn*
No. of deaths	4	9	19
Total numbers present when a death occurred	178	214	145

TABLE II. Chi-square analysis

Genotype	χ^2	d.f.	P
enen-Enen	.638	1	.5 — .3
EnEn	15.427	1	<.001
Total	16.065	2	<.001

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MANIFOLD EFFECTS OF THE GENE bi IN THE TOMATO

A. B. BURDICK AND T. R. MERTENS*

THREE INFLORESCENCE GENES
Figure 9

A—The gene *f* causes a ribbed, fasciated fruit, noticeable even in these immature fruits. *B*—Leafy inflorescence, *lf*, and jointless pedicel, *j*, act as one gene with two effects. *C*—Inflorescence with *f* and *lf j*. *D*— Bifurcate inflorescence, *bi*, usually has only one, but may have two or more dichotomies. *E*—Almost all stocks of *f* also carry *bi*. Many garden varieties are probably *bi f* but also have genes for smooth or non-ribbed fruits and an inhibitor of *bi*. *F*—The expression of *bi* in a *lf j* inflorescence is easily recognized.

THE recessive gene *bi*, bifurcate inflorescence, causes the inflorescences of a tomato plant to branch at least once so that, instead of being a simple raceme, the inflorescence is cymose. Figure 9*A* shows a normal racemose inflorescence. Figure 9*D* shows the branched inflorescence of a plant homozygous for *bi*.

This ostensibly simple genetic situation was only recently described[a], although branched types have long been familiar to tomato geneticists and breeders. The reasons why previous analyses may have failed will be evident from the unusual and interesting effects of this gene in certain genotypes.

The Rosette-Type Plant

Among the F_2 progeny of a Waltham Forcing × *d-f-j-lf-H-al-dm-wt* cross there were found two extremely fasciated plants, one of which is shown in Figures 10*E* (top view) and 10*F* (side view, defoliated). These rosette-type plants were fertile and produced 100 percent rosette-type offspring. (Dr. L. Butler, in a personal letter, states that such plants were observed many years ago by J. W. MacArthur.)

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MORPHOLOGICAL DISORDER

Figure 10

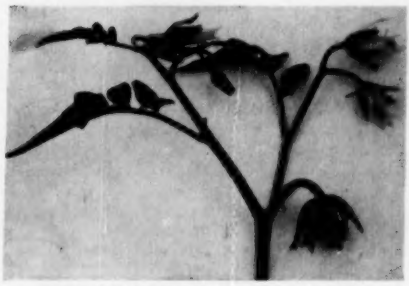
When *bi*, *f*, and *lf j* are all homozygous, the entire plant is affected. *A*—Normal plant. *B*—Rosette-type (*bi f lf j*) plant at about eight weeks of age. *C*—Rosette-type at nine weeks showing the much-fasciated single terminal flower. *D*—Top view of terminal flower at 10 weeks showing the burst of inflorescences and leaves just below. *E*—Top view of a very compact rosette-type plant. *F*—Side view of the plant in *E* after it had been defoliated to show stem fasciation.

With regard to growth habit, the rosette-type plant starts out as a normal seedling. The first noticeable departure from normal morphology occurs at about six weeks of age when normal plants develop the first inflorescence, usually after the tenth leaf (range, seventh to 14th leaf). Rosette-types fail to produce this first inflorescence, but continue to initiate leaves from an increasingly fasciated main axis until about 40 (range, 28-70) leaves have appeared. The main axis terminates in an extremely fasciated single flower as shown in Figure 10*C*. This flower seldom blooms, but about two weeks after its initial appearance a profusion of flowers and leaves emerge from the many leaf-axils just beneath it (Figure 10*D*).

At about the time the terminal flower appears, there is noticeable lateral branch development in the area where the first inflorescence should have been, i.e. at about the tenth leaf. These laterals develop without noticeable fasciation and produce inflorescences in the usual places. These inflorescences are leafy (*lf, lf*), the flowers have jointless pedicels (*j, j*), and the fruits are fasciated (*f, f*). In addition, the inflorescences are branched, as shown in Figure 11, with the first flower arising at the point of dichotomy.

Identification of *bi*

This branching, unusual for a *lf-j-f* inflorescence, appears in rosette-type × normal F_2 and backcross generations. Table I shows segregation and recombination values for *bi*, *lf j* (*lf j* does not recombine, therefore may be considered one gene) and *f* taken from a cross of rosette-type × red cherry hybrid. (Red cherry hybrid is an F_1 × 7, of line O14 and O18. Line O14 is a *p-r-y-d* tester with medium sized, two loculed fruits and wild-type inflorescence. Line O18 is our standard wild-type, red cherry, with small, two loculed fruits and wild-type inflorescence. Therefore, this hybrid serves as a wild-type with respect to the rosette-type genes in question). In this cross, *bi* segregates in the manner expected of



OUT OF CHAOS, THREE GENES
Figure 11

Lateral branches of rosette-type plants are usually normal. They produce inflorescences like the above, showing *bi*, *f*, and *lf j*.

a recessive gene (although *lf j* does not.) However, *bi* appears to be closely linked to *f* with only about 6.5 percent crossing-over. The genes *f* and *lf j* are known to be in linkage group V about 55 units apart. This distance is too large to allow determination of whether *bi* is to the left or right of *f*. Such information must come from a cross involving the gene *a* which lies 23 units to the right of *f*.

If the gene *bi*, proposed on the basis of the above studies of rosette-type segregation, is the gene usually responsible for branched inflorescence in the tomato then it should segregate in other material. To test this, the variety Criollo (J. A. Jenkins) which has branched inflorescence and fasciated fruit was crossed with the complete normal or wild-type, red cherry. The F_1 was completely wild-type. The results in F_2 are shown in Table II and, although the number of plants was small, the data support the identity of *bi* and its close linkage with *f*.

Another cross gave results that appear to conflict with the above. Criollo was crossed with Sutton's-Best-of-All, which is a large fruited, non-branched type. The F_1 of this cross was branched, indicating that Sutton's-Best must be *bi bi* but inhibited by an unknown recessive gene or genes. A small F_2 from this cross gave about equal numbers of *bi* and normals, also about equal numbers of *f* and normals.

These and other similar crosses require further investigation. It appears, however, that both *f* and *bi* are present although inhibited in some garden varieties, but the genetic nature of the inhibition is not clear.

Discussion

Figure 9*D-F* shows, respectively, *bi* alone, *bi* with *f* and *bi* with *lf j*. None of these types shows any alteration of stem morphology or inflorescence placement. However, when all three genes are homozygous, the plant is grossly changed (Figure 10*B-F*). The upper part of the main axis is highly fasciated, terminating in a single sunflower-like flower. Inflorescences do not appear on the main axis in spite of the fact that it has as many as 70 leaves.

Mertens and Burdick[3] have reported that rosette-type plants have not only linear and multiradiate fasciation but quite frequently show the unusual ring fasciation.

Each of these genes causes a certain amount of what might be called fasciation in the inflorescence. The gene *f* fasciates the fruit; *lf j* proliferates the inflorescence axis and *bi* divides it. The three genes together, however, do more than would be expected from a summation of their individual or two by two effects. This interaction indicates that each of them is associated with the production of a fasciation substance, call it fascinin, which, when present in sufficient quantity, fasciates other parts of the plant.

Additional evidence as to the mode of action of fascinin is available. Gorter[2] shows that ring fasciations can be induced in the variety Ailsa Craig by 2.3.5-triiodobenzoic acid (TIBA) treatment. The effect of TIBA is probably the result of inhibition of auxin transport from the meristem[4] since it is not possible to show that any less auxin is pro-

TABLE I. Segregation and recombination of *f*, *lf*, *j*, and *bi* in backcross and F_2 of rosette-type × red cherry hybrid

	Segregation							
	F_2 (1953 & 1954)				Backcross (1954)			
	Dom.	Rec.	P_1	P_2	Dom.	Rec.	P_3	P_4
bi	270	92	.80	.20	21	12	.10	.80
f	270	92	.80	.20	24	29	.20	.80
lf j	297	65	.01**	.50	26	27	.80	.50

P_1=Chi-square probability based on 3:1.
P_2=Heterogeneity chi-square probability based on two years.
P_3=Chi-square probability based on 1:1.
P_4=Heterogeneity chi-square probability based on two backcrosses.
**Highly significant.

Region*	Recombination Percent F_2 1953	F_2 1954	Total F_2	BC 1954	Map Distance†
bi-f	4.0	10.7	6.8	6.0	—
f-lf j	43.5	32.4	38.4	49.1	55.5
bi-lf j	48.2	28.3	38.4	47.2	—
No.	193	169	362	53	

lf j is considered one gene.
†Map by Butler.[1]

TABLE II. F_2 segregation of *bi* and *f* from the cross EB* (*bi bi f f* × + + + +)

Phenotype	Number
+ +	46
+ *f*	3
bi +	1
bi f	16
	Recombination = 5.26 ± 2.76%

*E = Line 089, Criollo, from J. A. Jenkins; B = Line 018, Red Cherry.

duced by a TIBA-treated plant. Therefore, the unknown plant substance, fascinin, may act as an inhibitor of auxin transport.

Literature Cited

1. Butler, L. The linkage map of the tomato. *Jour. Hered.* 43:25-35. 1952.

2. Gorter, C. J. The influence of 2.3.5-triiodobenzoic acid on the growing points of tomatoes. II. The initiation of ringfasciations. *Roy. Neth. Acad. Sci. Proc.* 54:181-190. 1951.

3. Mertens, T. R. and A. B. Burdick. The morphology, anatomy, and genetics of a stem fasciation in *Lycopersicon esculentum*. *Amer. Jour. Bot.* 41:726-732. 1954.

4. Thimann, K. V. and W. D. Bonner. The action of triiodobenzoic acid on growth. *Plant Physiol.* 23:158-161. 1948.

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A BALANCED STOCK WITH TRIPLE EYE-COLOR ALLELES IN THE WASP MORMONIELLA

A SERIES of multiple alleles for various eye colors, all recessive to wild-type brown, has been produced by mutation in the chalcidoid wasp Mormoniella. Several of these are isoalleles determining traits phenotypically similar, genotypically different. Some determine scarlet eye color, others oyster white. Different alleles are designated by letters or numbers following the name of the trait.

If scarlet-DR is crossed with oyster-DR, the compound females are wild-type brown which, unmated, produce only scarlet and oyster (haploid) sons. Scarlet-DR and oyster-DR are called complementary alleles showing codominance. Each is dominant over the other for a different factor, the compound females being double dominants. If scarlet-DR is crossed with oyster-250.6, it acts as a simple dominant and the scarlet daughters, unmated, produce only scarlet and oyster sons. These relationships may be expressed by postulating two elements in the wild-type allele, *O.S*, either of which may mutate. Scarlet-DR is then *O.st*, oyster-DR is *oy.S*, oyster-250.6 is double recessive, *oy-st*. Different types of mutation may occur in either element giving different eye colors such as dahlia, *da.S*, and mahogany, *O.mh*.

Several scarlet-lethals, *stl*-474 for example, have been obtained with recessive lethal effect and hence inviable in the male. These may be represented as *O.st.l*, the gene thus having a third element (or a third function). Oyster-423 is highly viable, *L*, but female-sterile, *fs*, and is doubly recessive for color, *oy.st*. The wild-type allele, *O.S.L.Fs*, therefore possesses four elements (or functions). A balanced stock is maintained, called *stl*-474/*oy*-423, in which females are scarlet (*O.st.l.Fs/oy-st.L.fs*) and oyster (*oy.st.L.fs/oy.st.L.fs*) in equal numbers and all males are oyster (*oy.st.L.fs*). The scarlet females of this stock are fertile, the oyster females sterile. The female-sterile males are apparently as fertile as other males.

A scarlet allele in this series, *st*-689, has been shown to cause female sterility similar to that of oyster-423. Crosses of scarlet-689 males with oyster-DR females result in wild-type daughters which are fertile producing scarlet and oyster sons only from unfertilized eggs. Crosses of scarlet-689 males with oyster-250.6 females result in scarlet daughters which are similarly fertile producing scarlet and oyster sons. Wild-type females heterozygous for scarlet-689 were crossed to oyster-423 males. The wild-type daughters were heterozygous for oyster-423 as expected. The scarlet daughters, *st*-689/*oy*-423, were sterile, *O.st.L.fs/oy.st.L.fs*, showing that the female-sterile factor is similar in the two alleles. When scarlet-689 males were crossed to *stl*-474/*oy*-423 females, all daughters were scarlet, half of them sterile (*O.st.l.Fs/oy.st.L.fs*) and half sterile (*oy.st.L.fs/O.st.L.fs*). Sons were all oyster and fertile (*oy.st.L.fs*).

A balanced stock was thus obtained with all females scarlet in every generation and with eye color of males alternating in successive generations. Settings of separate females of this stock demonstrate that half are sterile. The sterile females sting and inactivate the fly pupae, feed on the pupal juices through coagulation tubes formed between the pupae and puparia and live for many days, laying no eggs. A fertile female produces sons of one eye color, similar to that of her father and opposite to that of her brothers. The stock may be maintained in active condition by setting a mass culture from each generation or the diapause larvae may be refrigerated for well over a year as with pure-breeding stocks.

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SPERMATHECA SHAPE VARIATION IN DROSOPHILA AFFINIS

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FEMALE *Drosophila* normally possess paired dorsal seminal receptacles called spermathecae, and the shape of these varies considerably from species to species within the genus (Sturtevant[4]). In a few cases differences of spermatheca shape have been reported to be useful for distinguishing females of closely related forms (Spieth[3], Weeks[5]). Dobzhansky[1] observed significant differences of spermatheca shape between *Drosophila melanogaster* of different mutant genotypes.

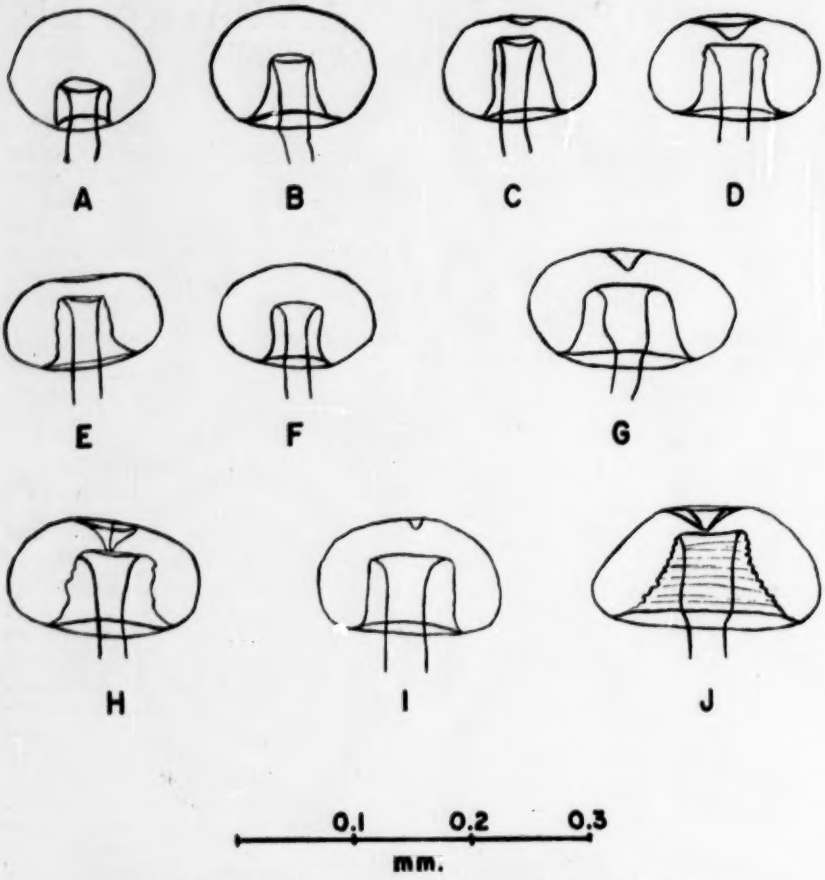
This study began as an attempt to find criteria for distinguishing between *Drosophila affinis* females and those of several closely related species. Laboratory strains of *D. affinis* (Florida, Illinois, Massachusetts, Nebraska, Tennessee, Texas), *D. algonquin* (Nebraska), *D. athabasca* (New York), *D. azteca* (Mexico), and *D. narragansett* (Massachusetts) were examined. Camera lucida drawings were prepared of spermathecae considered representative of the strains (Figure 12). It become apparent that *D. affinis* is rather variable as to spermatheca shape, and, although each of the other species was represented by one strain only, it seemed likely that, with the possible exception of *D. narragansett*, the other species were similarly variable. This variation involved the outside proportions of the chitinous capsule, the degree of penetration of the tube into the capsule, the occurrence of a terminal depression, and the nature of this indentation when present. It was not possible to find an altogether reliable criterion for distinguishing spermathecae of *D. affinis* from those of most of the other species. Only *D. narragansett*, with its peculiar broad tube invagination and lined terminal depression (Figure 12*J*), had a spermatheca shape sufficiently distinctive to afford easy recognition.

Among the strains of *D. affinis*, those from Florida (St. Petersburg) and Texas (Alice) showed the greatest difference as to spermatheca shape. The Florida spermathecae (Figure 13*A*) tended to be rather broad and flat, to have relatively much penetration of the tube inside the capsule, and usually had a more or less prominent terminal indentation. The Texas spermathecae (Figure 13*B*) tended to approach a spherical shape, to have a rather small tube penetration, and were never seen with a terminal indentation. Although there was some intrastrain variability, there was not much overlapping of spermatheca shape between the Florida and Texas strains. The other strains, on the other hand, generally had intermediate shapes and overlapped each other considerably. It was decided to investigate the influence on spermatheca shape of crosses involving the Florida and Texas strains. The following is an account of this investigation.

Procedure

It was planned to examine as many as possible of the spermathecae of females derived from a pair mating of each

*Contribution No. 283 of the Department of Zoology and Anatomy of the University of Nebraska. The author wishes to thank the following for having furnished *Drosophila* strains used in this study. Professor H. D. Stalker of Washington University (St. Louis) collected the single *D. affinis* female from which the Florida strain of this species was established. Professor A. H. Sturtevant (California Institute of Technology) supplied the Texas strain of *D. affinis*, as well as the strains of this species from Massachusetts and Tennessee. Other strains of *D. affinis* and related species were furnished by Professor Th. Dobzhansky (Columbia University), Professor P. T. Ives (Amherst College), Professor E. Mayr (Harvard University), Professor G. Mickey (Northwestern University), Professor W. P. Spencer (College of Wooster) and Dr. B. Wallace (Long Island Biological Association). The author is indebted to Professor H. W. Manter of the University of Nebraska for the photomicrographs of Figure 2.



SPERMATHECAE OF *D. AFFINIS* AND RELATED SPECIES

Figure 12

Camera lucida drawings of spermathecae of *Drosophila affinis* and several closely related species (the gelatinous, cellular envelope of the capsule is not illustrated). *A-F—D. affinis* (*A*—Texas, *B*—Tennessee, *C*—Nebraska, *D*—Florida, *E*—Illinois, and *F*—Massachusetts). *G—D. algonquin* (Nebraska). *H—D. athabasca* (New York). *I—D. azteca* (Mexico). *J—D. narragansett* (Massachusetts).

of the following kinds: Florida × Florida, Texas × Texas, both reciprocal crosses between Florida and Texas, $F_1 \times F_1$ matings following both reciprocal crosses, and all the possible backcrosses between the F_1 generations and the parent strains. Each mating was made in a culture bottle with standard *Drosophila* medium (corn meal—molasses—agar—Tegosept—yeast) and kept in an incubator at about 23.5°C (±1.5°). After eclosion (always within four days) female offspring were etherized and dissected in Ringer's Solution and the lower reproductive tract removed from each and mounted for microscopic observation. Rows of these reproductive tracts were scanned microscopically with the

aid of a mechanical stage. A single spermatheca was selected from each reproductive tract for observation and measurement. It was decided to select the first one of each pair encountered while scanning along the row. However, because of the need of a favorable orientation for making measurements (i.e. so as to present a lateral view), frequently one had to be taken regardless of position or else both had to be rejected. Measurements were made with an eyepiece micrometer, each unit of which corresponded to about 0.0032 mm. at the magnification used (ca. 200×). The following dimensions of the chitinous capsule of the spermathecae were measured: the width (a), the height (b), and the distance of penetration of the tube (c). Each spermatheca was also checked for the presence of a terminal depression, and if one was found, this was classed arbitrarily as small (Figure 12*C* and *E*), medium (Figure 12*D*), or large (Figure 13*A*; as here, terminal depressions classed as large often penetrated the opening of the tube inside the capsule).

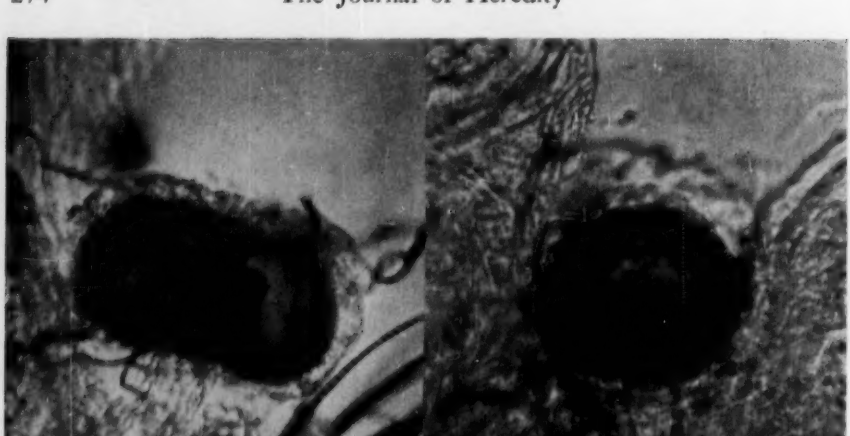
Tables I, II and III were prepared from the data accumulated from the progenies of the pair matings representing the various crosses. The numbers of individuals (N) reflect the different fecundities of the pair matings and the numbers of spermathecae recovered from the progenies. Table I presents the means of the width and height dimensions and the ratio of these (a/b), and Table II the means of the height dimension and the distance of tube penetration and the ratio of these (b/c). Table III shows the variation regarding the terminal depression.

Discussion of Results

The tables show that the progenies of the Florida × Florida and the Texas × Texas matings were quite distinct as to spermatheca shape. These progenies differed significantly (P less than 5 percent) as to the means of the a/b and b/c ratios (according to the t test, which was also applied to other differences of means), and the terminal indentation was generally present in the Florida spermathecae, regularly absent in those from Texas. However, there was some overlapping; the ranges of both ratios overlapped slightly, and two out of the 46 spermathecae of the Florida

TABLE I. ***D. affinis*** **Florida (St. Petersburg) and Texas (Alice) and generations derived from crossing these; each progeny obtained from a pair mating. Spermatheca width (a) and height (b) means; a/b ratio means (with standard errors), limits (in parentheses beneath means), and standard deviations (with standard errors)**

Parents	N	Mean a	Mean b	a/b $\bar{x}$	a/b s
Florida × Florida	46	22.0	13.9	1.59±0.07 (1.33—2.00)	0.50±0.05
Texas × Texas	73	18.3	15.1	1.22±0.01 (1.06—1.39)	0.07±0.01
Fla.♀ × Tex.♂	55	21.1	13.7	1.55±0.02 (1.33—1.81)	0.12±0.01
Tex.♀ × Fla.♂	62	21.2	14.3	1.49±0.02 (1.19—1.69)	0.14±0.01
F_1 (Fla.♀ × Tex.♂)	69	20.0	13.7	1.47±0.02 (1.19—1.77)	0.15±0.01
F_1 (Tex.♀ × Fla.♂)	40	20.6	14.2	1.47±0.03 (1.12—1.83)	0.17±0.02
F_1 (F.♀ × T.♂)♀ × F.♂	66	21.8	13.2	1.66±0.01 (1.40—1.92)	0.11±0.01
F.♀ × F_1 (F.♀ × T.♂)♂	39	22.0	13.9	1.58±0.02 (1.31—1.92)	0.14±0.02
F_1 (T.♀ × F.♂)♀ × F.♂	64	22.0	13.6	1.62±0.01 (1.33—1.92)	0.12±0.01
F.♀ × F_1 (T.♀ × F.♂)♂	20	22.1	13.2	1.69±0.03 (1.50—1.92)	0.14±0.02
F_1 (T.♀ × F.♂)♀ × T.♂	59	19.6	14.1	1.40±0.02 (1.13—1.75)	0.15±0.01
T.♀ × F_1 (T.♀ × F.♂)♂	32	19.0	14.5	1.32±0.03 (1.06—1.83)	0.17±0.02
F_1 (F.♀ × T.♂)♀ × T.♂	65	19.6	14.4	1.38±0.02 (1.00—1.75)	0.18±0.02
T.♀ × F_1 (F.♀ × T.♂)♂	60	19.2	14.5	1.31±0.02 (1.13—1.62)	0.11±0.01



***D. AFFINIS* SPERMATHECAE**

Figure 13

A and *B* shows photomicrographs of spermathecae of *D. affinis*: *A*—Florida (St. Petersburg); *B*—Texas (Alice).

progeny were without a terminal indentation.

The progenies resulting from crossing the Florida and Texas strains and the generations derived from these show the influence of hereditary factors contributed by both strains. However, the rather continuous nature of the variation and the considerable overlapping of the spermatheca shapes of the different generations make it difficult to determine the kinds of factors involved. The situation is probably also complicated by genetic heterogeneity of the Florida and Texas strains. Nevertheless, certain tentative conclusions may be drawn from the available data. These are indicated to a large extent by the general pattern of spermatheca shape variation as revealed by the a/b and b/c ratios and the frequencies of terminal indentations of the several kinds; as is pointed out below, differences of ratio means and of frequencies of depressions, though sometimes suggestive, are not always statistically significant.

The F_1 generations were characterized by intermediate means of the a/b and b/c ratios (Tables II and III), but closer to the corresponding Florida means than to those of Texas. In fact, the a/b ratio mean of the progeny of the Florida female mated to a Texas male was not significantly different from the Florida mean of this kind, although the other F_1 ratio means were significantly different from the corresponding means of the parent strains. Regarding both ratio means, each F_1 was somewhat closer than the other one to the progeny representing the strain of its female parent, and the differences are statistically significant (less complete data were responsible for the report of Miller[2] of no influence of direction of cross). The F_2 means of the a/b and b/c ratios were shifted in the direction of the corresponding Texas values (though only the Florida female × Texas male F_1-F_2 differences were significant), and the two F_2 generations were alike as to these means. The backcrosses to the Florida and Texas strains gave progenies with means of the a/b and b/c ratios invariably displaced in the directions of the strains used in backcrossing, especially those to the Florida strain. The backcrosses to Texas (but not to Florida) showed slightly greater shifts of ratio means toward the parental strain where a female of this strain was used in backcrossing, but these differences are not significant. It may be concluded that Florida factors influencing the a/b and b/c ratios show some dominance over those of Texas. In addition, the difference between the F_1 progenies and those gotten from backcrosses to Texas suggest a possible maternal effect on spermatheca shape.

Regarding the terminal indentation of the spermatheca (Table III), the F_1 generations were intermediate to Florida and Texas, but more similar to Texas than to Florida. The F_2 generations were still more similar to Texas than the F_1's, but application of the chi-square test to the frequencies of sperma-

TABLE II. ***D. affinis*** **Florida (St. Petersburg) and Texas (Alice) and generations derived from crossing these; each progeny obtained from a pair mating. Spermatheca height (b) and distance of tube penetration (c) means; b/c ratio means (with standard errors), limits (in parentheses beneath means), and standard deviations (with standard errors)**

Parents	N	Mean b	Mean c	b/c $\bar{x}$	b/c s
Florida × Florida	46	13.9	8.9	1.58±0.02 (1.30—2.00)	0.10±0.01
Texas × Texas	73	15.1	5.5	2.84±0.07 (2.00—5.33)	0.60±0.05
Fla.♀ × Tex.♂	55	13.7	8.3	1.66±0.01 (1.30—2.50)	0.21±0.02
Tex.♀ × Fla.♂	62	14.3	7.6	1.90±0.03 (1.60—2.50)	0.22±0.02
F_1 (Fla.♀ × Tex.♂)	69	13.7	7.2	1.96±0.05 (1.44—3.20)	0.40±0.04
F_1 (Tex.♀ × Fla.♂)	40	14.2	7.5	1.96±0.08 (1.44—3.40)	0.49±0.06
F_1 (F.♀ × T.♂)♀ × F.♂	66	13.2	8.8	1.51±0.02 (1.20—2.00)	0.16±0.01
F.♀ × F_1 (F.♀ × T.♂)♂	39	13.9	8.4	1.67±0.02 (1.40—2.00)	0.13±0.01
F_1 (T.♀ × F.♂)♀ × F.♂	64	13.6	8.8	1.56±0.02 (1.30—2.00)	0.15±0.01
F.♀ × F_1 (T.♀ × F.♂)♂	20	13.2	8.2	1.62±0.02 (1.44—1.86)	0.10±0.02
F_1 (T.♀ × F.♂)♀ × T.♂	59	14.1	6.6	2.25±0.10 (1.44—5.33)	0.74±0.07
T.♀ × F_1 (T.♀ × F.♂)♂	32	14.5	6.2	2.46±0.11 (1.50—3.75)	0.62±0.08
F_1 (F.♀ × T.♂)♀ × T.♂	65	14.4	6.8	2.22±0.07 (1.44—3.75)	0.56±0.05
T.♀ × F_1 (F.♀ × T.♂)♂	60	14.5	6.3	2.41±0.07 (1.56—3.75)	0.51±0.05

TABLE III. ***D. affinis*** **Florida (St. Petersburg) and Texas (Alice) and generations derived from crossing these; each progeny obtained from a pair mating. Numbers and percentages (in parentheses beneath) of spermathecae with no terminal indentation and with terminal indentations classed as small, medium, and large**

Parents	N	Indentation None	Small	Med.	Large
Florida × Florida	46	2 (4.3)	3 (6.5)	14 (30.4)	27 (58.7)
Texas × Texas	73	73 (100)	0	0	0
Fla.♀ × Tex.♂	55	42 (76.4)	13 (23.6)	0	0
Tex.♀ × Fla.♂	62	50 (80.6)	10 (16.1)	2 (3.2)	0
F_1 (Fla.♀ × Tex.♂)	69	68 (98.6)	1 (1.4)	0	0
F_1 (Tex.♀ × Fla.♂)	40	37 (92.5)	2 (5.0)	1 (2.5)	0
F_1 (F.♀ × T.♂)♀ × F.♂	66	7 (10.6)	31 (47.0)	15 (22.7)	13 (19.7)
F.♀ × F_1 (F.♀ × T.♂)♂	39	6 (15.4)	11 (28.2)	7 (17.9)	15 (38.5)
F_1 (T.♀ × F.♂)♀ × F.♂	64	11 (17.2)	18 (28.1)	18 (28.1)	17 (26.6)
F.♀ × F_1 (T.♀ × F.♂)♂	20	3 (15.0)	8 (40.0)	6 (30.0)	3 (15.0)
F_1 (T.♀ × F.♂)♀ × T.♂	59	57 (96.6)	2 (3.4)	0	0
T.♀ × F_1 (T.♀ × F.♂)♂	32	32 (100)	0	0	0
F_1 (F.♀ × T.♂)♀ × T.♂	65	59 (90.8)	4 (6.2)	2 (3.1)	0
T.♀ × F_1 (F.♀ × T.♂)♂	60	60 (100)	0	0	0

thecae with and without indentations (i.e. by combining the different classes of depressions) shows only the shift of the Florida female × Texas male F_2 to be statistically significant. The backcrosses to Florida and Texas regularly produced offspring resembling the strain used in backcrossing, especially the backcrosses to Texas. Some dominance of Texas factors preventing terminal indentations is suggested by the F_1 generations and the more complete return to the parental type accompanying the Texas backcrosess. The fact that the F_2 progenies resembled Texas more than did the F_1's (contrary to the expectation based on Texas dominance) may be due to a dependence of terminal indentation formation on other aspects of spermatheca shape (as indicated by the a/b and b/c ratios) under the influence of somewhat dominant Florida factors.

Conclusions

D. affinis may be described as polymorphic as to spermatheca shape, with genetic factors contributing to this polymorphism. A rather complex genetic basis for spermatheca shape variation seems likely, although the possibility of a small number of major factors with variable effects cannot be excluded.

The dissimilarity of the Florida and Texas strains suggests that which sometimes exists between different taxonomic groups—e.g. subspecies, even species. However, there was no detectable reproductive isolation between these strains (e.g. their hybrids were fully fertile), and, moreover, the various other strains of *D. affinis* exhibited spermatheca shapes of a more or less intermediate nature and varied so as to overlap each other broadly. The available strains do not suggest a very simple geographical distribution of spermatheca shape variation. It is, of course, not possible to conclude whether any of the strains are very representative of the wild populations from which they originated. It would be especially desirable to investigate further the variation of spermatheca shape in southern Florida and southern Texas populations of *D. affinis*.

Summary

Spermatheca shape was investigated in *Drosophila affinis* and several similar species (*D. algonquin, athabasca, azteca,* and *narragansett*). With the exception of *D. narragansett,* none of these other species had spermathecae easily distinguishable from those of *D. affinis*. Considerable intraspecific variation in spermatheca shape was revealed within *D. affinis*. Laboratory strains of *D. affinis* from Florida and Texas were found to be especially different in this respect. The Florida spermathecae tended to be rather broad and flat, to have relatively much penetration of the tube inside the capsule, and usually had a more or less prominent terminal indentation. The Texas spermathecae tended to approach a spherical shape, to have rather little tube penetration, and these were never found to have a terminal indentation. Crosses between the Florida and Texas strains and the offspring of these yielded progenies with more or less intermediate spermatheca shapes, with some evidence of dominance of both Florida and Texas factors and of a slight maternal effect.

Literature Cited

1. Dobzhansky, Th. Über den Bau des Geschlechtsapparats einiger Mutanten von *Drosophila melanogaster*. *Zeits, indukt. Abst. u. Vererbungsl.* 34:245-248. 1924.

2. Miller, D. D. Intraspecific variation in spermatheca morphology in *Drosophila affinis* Sturtevant. *Rec. of the Genet. Soc. Am.* 23: 56. 1954.

3. Spieth, H. T. Sexual behavior and isolation in *Drosophila*. II. The interspecific mating behavior of species of the *willistoni* group. *Evolution* III(1):67-81. 1949.

4. Sturtevant, A. H. The North American Species of *Drosophila*. 150 pp. The Carnegie Institution of Washington. 1921.

5. Weeks, L. Studies on the two subspecies, *D. melanica melanica* and *D. melanica paramelanica*. *Drosophila Information Service* 27:118-119. 1953.

MALE INFLUENCE ON SEX RATIO OF OFFSPRING IN HIGH AND LOW BLOOD-pH

Lines of Mice

J. A. WEIR*

THE basic theory of sex determination indicates that the two sexes should be produced in equal numbers; and observed departures from such equality have given rise to a vast literature. Guesswork, based on knowledge that segregation of X and Y chromosomes provides the mechanism for production of equal numbers of males and females, leads to the premise that the two kinds of sperm are regularly deposited in the female tract in equal numbers (a direct experimental approach is vitiated by technical difficulties). In theory, the environment of the female ducts may exert a selective influence so that, apart from a possible intrinsic difference between the fertilizing capacity of X- and Y-sperm, the male should have little influence on the primary sex ratio. Contrary to this view, evidence will be presented from mice, which suggests that conditions in some part of the male reproductive tract may affect viability of X or Y sperm, or that the two kinds of sperm may not be produced in equal numbers.

It was suggested in an earlier communication[1,2] that the observed association of an excess of males with a high blood-pH and of an excess of females with a low blood-pH might have been due to conditions in the dam's reproductive tract, in which cervical mucus with a relatively high pH favored Y-bearing sperm and that with a relatively low pH favored X-bearing sperm. According to evidence based on data from reciprocal crosses (presented below) this hypothesis must be abandoned. It is now clear that the sire must be held accountable for the characteristic sex ratios of litters from pHH and pHL lines of mice. This is in contrast to King's findings for rats,[7] which showed for outcrosses that it was the female parent which largely determined the characteristic sex ratio of each of the different inbred lines.

A search of the literature has disclosed one case involving untreated animals in which a male influence on the sex ratio in mice has been claimed. Gates[3] tested for the possible linkage of the waltzing factor with other factors in the mouse and in the process kept males in continuous service for periods up to 13 months. Eight males (genotype *p*/+, *d*/*d*, *s*/*s*, *a*/*a*, *b*/*b*) sired a total of 1437 mice with progeny per sire ranging from 41 to 570. The sex ratio in the polygynous series did not differ significantly from that of the average for the colony indicating no apparent influence of mating frequency on the sex ratio. Gates suggested that the high and low ratios represented individual characteristics of certain males although the differences certainly were not large. The sex ratios for the eight progeny groups represented in Gates' table 1, recalculated as percent males with standard errors, turn out to be: 51.9 ± 2.1, 49.5 ± 3.0, 48.3 ± 3.0, 44.9 ± 5.6, 45.4 ± 5.7, 45.4 ± 6.2, 45.0 ± 7.1, 56.1 ± 7.8.

Howard *et al.*[5] found no evidence for genetic factors acting through the male or female parents of reciprocal crosses and Falconer's[2] selection experiment gave no evidence of genetic variation of the sex ratio in the outbred population of mice employed. The results to be presented, which confirm and extend the

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preliminary findings,[13] may represent an unusual if not unique situation in mice.

Materials and Methods

The strains of mice, which have been previously described[12, 14, 15] were the outbred T (sex ratio 50 percent males), pHH (high blood-pH, sex ratio 55 percent males), and pHL (low blood-pH, sex ratio 45 percent males). A test for the effect of male or female parent on sex ratio of the progeny simply requires use of reciprocal crosses between high and low sex ratio lines. However, the capricious nature of sex ratios in mice makes hazardous the drawing of conclusions from single experiments.[6, 11] The following precautions have been taken: (1) Mated pairs have been left together throughout their entire reproductive span. (2) Matings have been made at intervals during any one year so first litters would arrive during different seasons. (3) The experiment has been repeated once in its entirety and first litters obtained from a third complete set of matings.

The breeding plan entailed making up single pair *inter se* and reciprocal cross matings. Animals used for crossing were drawn from the same litters as those used for full sib matings and all were mated at the same time in any one series so environmental effects would not be confounded with type of mating. The practice of leaving pairs together throughout their entire reproductive span reduced the number of new matings that could be accommodated by the available space but provided data for evaluation of age, parity, season, and year. Results for the years 1953, 1954 and 1955 will be presented separately. Since the results from replication are in essential agreement with respect to male influence on the sex ratio it seems unlikely that further data will add new matter or require serious modification of the conclusions drawn. Concerning the role of non-genetic influences on the sex ratio it seems appropriate in this paper merely to indicate the sources of variation and to defer an attempt to analyze their nature.

Thirty-day sex ratios were used in Series I and II but in the final series, pHH, pHL, and T litters were sexed at birth as well as at weaning.

TABLE I. Series I. Weaning sex ratios in successive litters of pHH and pHL mice

Litter Order	No. of Litters	♀♀	♂♂	Total	% ♂♂	Standard Error of Sex Ratio	Mean Litter Size
				pHH			
1	20	67	43	110	39.1	4.8	5.5
2	17	47	54	101	53.5	5.0	5.9
3	10	28	38	66	57.6	6.2	6.6
4	1	9	13	22			
5	2	8	5	13	52.5	7.9	6.7
6	1	2	3	5			
	51	161	156	317	49.2	2.8	6.0
				pHL			
1	26	94	56	150	37.3	4.1	5.8
2	24	79	48	127	38.5	4.4	5.3
3	19	51	35	86	40.7	5.4	4.5
4	13	33	26	59	44.1	6.5	4.5
5	9	17	15	32	46.9	8.8	3.6
6	4	8	8	16			
7	2	6	3	9			
8	1	1	2	3	40.6	8.8	4.0
9	1	4	0	4			
	99	293	193	486	39.7	2.3	4.9

Experimental Results

1. Series I—1953 Matings

Life time production of pHH and pHL matings of 1953-54 are shown in Table I. These matings served as controls for the outcross matings. Comparing total progeny from 53 pHH litters and 99 pHL litters the sex ratio picture presents a repetition of what has become commonplace, namely, a stable sex ratio difference of approximately 10 percent between strains. The actual sex ratios of 49.2±2.8 and 39.7±2.3 are comparable to the ratios of the first selected generation, of 1949, in which 50.5± 3.5 percent males occurred in pHH litters and 41.2±3.8 percent males in pHL.[12] But, since the characteristic sex ratio of the two strains had been hitherto expressed in first litters, the occurrence of a significant excess of females in first litters of pHH requires an explanation. Litters were always enumerated and usually sexed soon after birth, and an excess of females was noted, but the sex classification was not permanently recorded until weaning. Post-natal mortality could not have seriously modified the sex ratio in any event since only five mice were lost between birth and weaning; i.e. if all five were males the birth sex ratio would have been 41.7 percent males compared to 39.1 percent at weaning. However, adumbrations of a differential total mortality effect, postnatal and prenatal, are indicated: (1) Litter size at weaning for first litters was smaller than litter size for succeeding litters, a situation which is common in mice, but inconsistent with the main body of data in the present study. (2) Postnatal

mortality may fall more heavily on the male in pHH mice (see Series III).

A search for additional influences on the first litter sex ratio proved fruitless. Age of parents was not a factor and temporary environmental influences did not seem too important since the female excess was not confined to one season. For example, there were 16 females to four males in five first litters born in April and 18 females to nine males in four first litters born the following January.

From results of reciprocal crosses of pHH and pHL (Table II), the influence of the male becomes immediately and convincingly apparent. There is even an indication of an exaggerated effect in crosses as compared to pure strain matings. Sex ratios were 62.6 ± 5.2 for pHL ♀ × pHH ♂, and 39.8 ± 4.8 for pHH ♀ × pHL ♂. Although discovery that the male parent determines the sex ratio of his offspring surely represents the most important outcome of the experiment, certain ancillary features, of a less clear cut nature, require an explanation.

Again, as in the pure strain matings, the pHH females (Table II) had smaller first litters than second or following litters. When it is recalled that the females used for *inter se* and outcross matings were sisters, and that in all other mating series (see also Tables III and IV) litter size declined after the first litter, the data again suggest a differential mortality effect. In other words, the occurrence of 23 females to nine males (28.1 percent males) in first litters of pHH ♀ × pHL ♂, might be interpreted as a result of the combined effects of an initial female surplus due to the pHL sire's contribution, and a subsequent loss of male embryos *in utero*.

Although the 1953-54 matings served to establish that the sex ratio difference between pHH and pHL strains of mice is determined almost entirely by influences which act through the male parent, repetition of the experiment was deemed necessary. Some of the questions remaining to be answered were: Would the low sex ratio in pHH first litters repeat? Would the exaggerated sex ratio difference in reciprocal crosses repeat?

2. Series II—1954 Matings

The sex ratio picture for the 1954 matings presents the same general features as the 1953 series, but with certain differences in detail. The pure strain sex ratios of 54.4 and 40.8 percent males for pHH and pHL mice were not significantly different from the ratios of the previous series. It will be observed that the low sex ratio and small litter size found in Series I for first litters from pHH females did not repeat in Series II. It seems, therefore, that the low sex ratio in Series I, which may be explicable on a basis of male mortality, does not represent a general phenomenon. At the same time, the apparent exaggeration of sex ratio differences in crosses, in the first series, seems to be nothing more than an accident of sampling. In Series II crosses, sex ratios were 52.6 percent males from pHL ♀ × pHH ♂ and 35.2 percent males from pHH ♀ × pHL ♂ compared to the pure strain ratios of 54.4 percent for pHH and 40.8 percent for pHL.

Apart from the progeny of pHH females in Series I, there seems to be no relationship between litter size and sex ratio. Falconer's experience (personal communication) has been similar but Howard *et al.*[5] found a significant rise in the proportion of males with increasing litter size for both inbreds and hybrids. Differential mortality, which will be discussed in a later section, does not seem to be a major consideration in the present study, but there are features of the fertility record which deserve comment at this point. It will be seen (Tables I-IV) that, with the exceptions already noted, litter size was at a maximum in

TABLE II. Series I. Weaning sex ratios in successive litters from reciprocal crosses

Litter Order	No. of Litters	♀♀	♂♂	Total	% ♂♂	Standard Error of Sex Ratio	Mean Litter Size
pHL ♀ × pHH ♂							
1	6	16	21	37	56.8	8.2	6.2
2	4	10	15	25	60.0	10.0	6.2
3	3	6	15	21	72.4	9.1	5.8
4	2	2	6	8			
	15	34	57	91	62.6	5.2	6.1
pHH ♀ × pHL ♂							
1	5	23	9	32	28.1	8.8	6.4
2	4	13	15	28	53.6	9.3	7.0
3	3	7	7	14			
4	3	9	6	15			
5	2	8	4	12	39.6	7.2	5.3
6	1	5	2	7			
	18	65	43	108	39.8	4.8	6.0

TABLE III. Series II. Weaning sex ratios in successive litters of pHH and pHL mice

Litter Order	No. of Litters	♀♀	♂♂	Total	% ♂♂	Standard Error of Sex Ratio	Mean Litter Size
pHH							
1	8	27	28	55	50.9	6.7	6.9
2	3	8	11	19	57.9	11.5	6.3
3	2	3	6	9	66.7	16.7	
4	1	3	4	7	57.1	18.9	
	14	41	49	90	54.4	5.3	6.4
pHL							
1	11	43	31	74	41.9	5.8	6.7
2	11	32	23	55	41.8	6.7	5.0
3	10	36	18	54	33.3	6.8	5.4
4	7	21	14	35	40.0	8.4	5.0
5	4	10	12	22	54.5	10.7	5.5
	43	142	98	240	40.8	3.2	5.6

first litters in both inbreds and F_1 litters. No particular significance need be attached to this observation but it is at variance with the general experience for mice. It will be noted that, during the period under consideration, pHH mice were superior from a standpoint of litter size, but that pHL mice produced more litters. There were also more sterile matings in the pHH strain. Dissections have revealed a high incidence of uterine cysts in older females of both strains whereas sperm from the ductus deferens of old males usually prove to be motile. The low fertility threshold seems therefore to be generally attributable to the female. It should be noted that a complete analysis of the fertility data would have to take into account age of parents and incidence of litters from post partum matings.

3. Series III—1955 Matings

In connection with artificial insemination experiments, matings were made up as before except that females were isolated when pregnant and allowed to produce only one litter. The mice were kept in a non air-conditioned room in which the light cycle was reversed; otherwise management of the colony was unchanged. Results of this series are here included because sex was classified at birth as well as at weaning. It is evident that postnatal mortality does not contribute significantly to the variation of sex ratios. Because the numbers are small, records from individual litters are included in Table V. Altogether the losses amounted to 13 females and 10 males out of 259 mice born. Expressed in terms of litters, there were seven litters in which one or more females were lost and eight litters in which one or more males were lost. It must be pointed out that the pHL litter of nine, in which six of seven females died, was abnormal and that one of the two males died after weaning. Weights of the three survivors at 34 days of age were: ♀—12 grams, ♂♂—8 and 9 grams (half the normal weight). Even though the limited data from sexing at birth do not permit a decision concerning the influence that differential mortality may exert on the sex ratio, it is clear that postnatal mortality could not account for any significant fraction of the total observed variation. The overall tendency is for more males than females to die (see also Tables I and II, *Journal of Heredity*, July-August, 1953).

TABLE IV. Series II. Weaning sex ratios in successive litters from reciprocal crosses

Litter Order	No. of Litters	♀♀	♂♂	Total	% ♂♂	Standard Error of Sex Ratio	Mean Litter Size
			pHL ♀ × pHH ♂				
1	6	25	23	48	47.9	7.2	8.0
2	5	17	15	32	46.9	8.8	6.4
3	3	6	11	17	64.7	12.1	5.7
4	2	4	9	11			
5	1	2	1	3	63.2	11.5	4.8
6	1	1	2	3			
	18	55	61	116	52.6	4.6	6.4
			pHH ♀ × pHL ♂				
1	7	28	27	55	49.1	6.7	7.8
2	7	31	10	41	24.4	7.8	5.8
3	3	18	3	21	14.3	10.9	7.0
4	2	4	4	8	50.0	17.7	4.0
	19	81	44	125	35.2	4.5	6.6

4. Matings of Outbred (T) Mice—1954-55

The outbred T strain, from which pHH and pHL strains were derived originally, provides especially good material for study of postnatal mortality as the proportion of sexes at weaning is equality (49.9 ± 1.0 percent males). Many investigators classify sex at birth, so it seems appropriate to provide comparable information.

The data of Table VI, which have been extracted from a larger study, the results of which will be presented elsewhere, include all litters which were classified both at birth and at weaning. First litters are not included in the table as none were classified at birth. Losses amounted to only three mice out of 164 born in 19 first litters. Sex ratio at weaning was 47.2 percent males. Most of the second litters were killed at birth and sexed by dissection (sex ratio 48.7 percent males), hence, the small number of second litters included in Table VI. The main body of data, from which the material included in Table VI has been extracted, included 652 mice in 85 litters, from 19 families, each family consisting of progeny of a single pair. Sex ratio at birth was 48.6 ± 2.0. It will be noted that this ratio does not differ significantly from equality of sexes so no importance can be attached to the "low" sex ratio for the 47 litters included in Table VI.

The total postnatal mortality, which amounted to 5.3 percent, might seem high for an outbred line of mice. However, the conditions encountered by the mice were not conducive to maximum survival. The parents were six months old when first mated and pairs were left together continuously, with the result that most of the litters were from post partum matings. It seems, therefore, that the four females and 14 males which failed to reach weaning age were victims of competition for survival under rigorous but natural conditions. No statistical test is necessary to assess the significance of the greater vulnerability of the male. It is apparent also that classification at weaning time provides sex ratio data which are not too different from those obtained from classification at birth. Classification at birth has the disadvantage that there must be frequent subsequent checks to eliminate errors.

The Problem of Differential Mortality

The problem of differential mortality is not directly applicable to the present investigation but the ensuing discussion is offered in recog-

nition of the fact that prenatal elimination provides the bulwark for most arguments pertaining to sex ratio theory. Differential mortality has not been completely ruled out in the present investigation, but it is difficult to devise a theoretical model by which an influence of the sire will be expressed in such a way that more male embryos will die when low blood-pH males are used and more female embryos will die when high blood-pH males are used. In the absence of direct proof, the law of parsimony would lead one to reject any general hypothesis which relies on differential mortality of embryos. It would seem as though we are actually dealing with variations in sex ratio which are basically traceable to events which occur before fertilization.

Great difficulties attend assessment of prenatal mortality in mammals, but there is reliable evidence that a considerable loss of embryos is not uncommon in polytocous species.[1,4] After fertilization there is a rather long period of development before the sexes can be classified, so there is always opportunity for prenatal mortality to influence the sex ratio. In theory, selective elimination presents the problem of thresholds, and what is needed is conclusive experimental evidence that, at a particular stage, or at successive stages, one sex actually is or is not eliminated at the expense of the other; such evidence is lacking.

There are ways in which the net effect of selective elimination has been estimated. For example, Parkes[10] found that under normal conditions fetal regression without abortion amounted to 10.8 per 100 full-time fetuses and in pregnancies following immediately on previous ones the amount was raised to as much as 23.1. There was a corresponding drop in sex ratio at birth from 54.1 percent males to 38.3 percent males leading to the inference that "pre-natal mortality falls preponderatingly on the males." On the other hand, Howard *et al.*[5] present convincing evidence from their data that the female fetus is the more vulnerable. Pregnant females were isolated from the males and not returned until the litters were four weeks old so the data are not comparable to those of Parkes.[10] Howard *et al.*[5] have been careful to point out that they have made no assumptions concerning the primary sex ratio.

Absence of prenatal differential mortality in mice was inferred by MacDowell and Lord[8,9] from data based on number of corpora lutea for each litter, and Weir,[12] from a compari-

TABLE V. Series III. Sex ratios and mean litter sizes in the inbreds and reciprocal crosses

Strain	Number of Litters	Classified at Birth: Proportion of Males	Sex Ratio	Standard Error of Sex Ratio	Litter Size	Classified at Weaning: Proportion of Males	Sex Ratio	Standard Error of Sex Ratio	Litter Size
pHH	10	6/9, 5/9, 4/6 3/5, 2/9, 2/6 2/8, 4/6, 3/6 4/4	51.5	6.1	6.8	5/8, 3/7, 4/6 3/5, 2/9, 2/6 2/8, 2/3, 3/6 4/4	48.4	6.4	6.2
pHL	8	1/7, 2/5, 2/3 *2/9, 2/8, 0/4 1/3, 0/5	22.7	7.5	5.5	1/7, 2/5, 2/3 2/3, 2/8, 0/3 1/3, 0/5	27.9	8.2	4.6
HL♀ × HH♂	8	3/7, 2/3, 3/5 6/9, 1/8, 5/10 1/6, 6/8	48.2	6.7	7.0	2/6, 2/3, 2/4 5/9, 1/6, 5/10 **1/5, 6/8**	48.0	7.1	6.2
pHH♀ × pHL♂	12	4/6, 3/6, 2/4 3/7, 3/10, 6/9 1/7, 4/10, 1/8 2/10, 3/9, 0/5	17.4	5.2	7.6	4/6, 3/6, 2/4 3/7, 2/8, 6/9 3/7, 3/9, 1/8 2/9, 3/9, 0/5	16.8	5.4	7.2

*Abnormal litter, six females died before weaning, one male died after weaning.

TABLE VI. Sex ratios of outbred (T) mice

Litter Order	Classified at Birth: Mean Litter Size	No. of Litters	♀♀	♂♂	Total Mice	Sex Ratio	Standard Error of Sex Ratio	Classified at Weaning: No. of Litters	♀♀	♂♂	Total Mice	Sex Ratio	Standard Error of Sex Ratio	Birth to Weaning Loss: ♀♀	♂♂
2	7.8	5	21	18	39	46.2	8.0	5	20	17	37	45.9	8.2	1	1
3	7.5	15	57	56	113	49.6	4.7	15	57	50	107	46.7	4.9	0	6
4	7.3	10	33	40	73	54.8	5.8	10	31	35	66	53.0	6.2	2	5
5	6.4	9	32	26	58	44.8	6.6	9	32	25	57	43.8	6.6	0	1
6	6.8	5	23	11	34	32.4	8.8	5	22	10	32	31.2	8.8	1	1
7	7.0	3	14	7	21	33.3	10.9	3	14	7	21	33.3	10.9	0	0
		47	180	158	338	46.7	2.7	47	176	144	320	45.0	2.8	4	14

son between data for litters with no birth-to-weaning loss and data which included all litters, concluded that postnatal mortality also was essentially non-selective. The evidence upon which the conclusions are based is not convincing in either instance, since it can be shown that extrapolation from secondary to primary sex ratios is not valid when there is no knowledge of the sex of mice which failed to survive. This may be stated in mathematical terms as follows:

Each egg fertilized results in one of three events; M, that it develops to a stage at which the sex can be determined and is found to be a male; F, that it develops to a stage at which the sex can be determined and is found to be a female; and U, that it fails to develop to a stage at which the sex can be determined.

Now, for a mating of given types and for total number, N, of eggs fertilized, suppose that each of the N eggs independently yields one of the events M, F, and U with probabilities p_1, p_2, and $1-(p_1+p_2)$ respectively. Let n_1 and n_2 denote the number of eggs yielding M and F, respectively. Then the probability distribution of (n_1, n_2) is clearly a multinomial distribution.

It is easily seen that

$$E\left(\frac{n_1}{n_1+n_2}\right) = \frac{p_1}{p_1+p_2} = \frac{1}{1+\frac{p_2}{p_1}}$$

since for every s, $0<s\leq N$, the conditional distribution of n_1 given that $n_1+n_2=s$ is the binomial with parameters s and $p_1/(p_1+p_2)$ and hence,

$$E\left(\frac{n_1}{n_1+n_2}\,\middle|\, n_1+n_2=s\right) = \frac{p_1}{p_1+p_2}$$

Note that $E(n_1/(n_1+n_2)$ depends only on the ratio p_2/p_1, so that even were it considered proper to assume that the probabilities are dependent on the number N, as long as the ratio p_2/p_1 does not vary with N the result holds.

The influence which differential mortality may exert on the sex ratio is difficult to assess but it is generally concluded that variability of secondary sex ratios is likely to be due both to differences in the primary sex ratio and to different frequencies of prenatal mortality. Experiments in progress, using the technique of artificial insemination, are directed toward elucidation of the nature of male influence on the primary sex ratio of pHH and pHL lines of mice.

Summary and Conclusions

Inter se and reciprocal cross matings of pHH and pHL mice were made, and, in two of the three series, pairs were left together until offspring ceased to be produced. In the first experiment, Series I, 1953, sex ratios, expressed as percent males at weaning were: pHH 49.2 ± 2.8; pHL 39.7 ± 2.3; pHL ♀ × pHH ♂ 62.6 ± 5.2; pHH ♀ × pHL ♂ 39.8 ± 4.8. First litters of pHH had a low sex ratio (39.1 percent males), which was independent of season or age of parents; it is suggested that in this isolated instance, males may have been lost *in utero*.

Repetition of the experiment, Series II, led to the same general results, viz. an overriding male influence in reciprocal crosses. Sex ratios were: pHH 54.4 ± 5.3; pHL 40.8 ± 3.2; pHL ♀ × pHH ♂ 52.6 ± 4.6; pHH ♀ × pHL ♂ 35.2 ± 4.5. Neither the "low" sex ratio of first litters of pHH nor the exaggerated sex ratio difference of Series I reciprocal crosses were repeated in Series II.

The third series, containing only first litters, had sex ratios at weaning as follows: pHH 48.4 ± 6.4; pHL 27.0 ± 8.2; pHL ♀ × pHH ♂ 48.0 ± 7.1; pHH ♀ × pHL ♂ 36.8 ± 5.4. Sex was also classified at birth. Except for one abnormal pHL litter, in which six females died before weaning, postnatal losses were small.

The overall tendency is for more males than females to die. For example, of 338 outbred (T) mice born, in 47 litters, four females and 14 males were lost between birth and weaning. Classification at birth does not seem to present any advantages other than provision of an opportunity to assess postnatal mortality.

The problem of differential prenatal mortality is discussed. In some mouse stocks the male fetus seems to be the more vulnerable; in others it is the female. On the other hand, by present methods, it does not seem possible to prove the absence of differential mortality in any particular mouse stock.

In the investigation, here reported, there is no evidence that prenatal mortality has any significant influence on the overall results. The sex ratio difference between pHH and pHL is wholly accountable by the influence of the sire. The physiological basis for the results is under investigation.

Literature Cited

1. Brambell, F. W. Rogers. Prenatal mortality in mammals. *Biol. Rev.* 23:370-407. 1948.

2. Falconer, D. S. Selection for sex ratio in mice and *Drosophila*. *Am. Nat.* 88:385-397. 1954.

3. Gates, W. H. The effect of polygyny on the sex ratio of mice (*Mus musculus*). *Jour. Exp. Biol.* 7:235-240. 1930.

4. Hammond, J. Fertility in mammals and birds. *Biol. Rev.* 16:165-190. 1941.

5. Howard, A., A. McLaren, D. Michie and G. Sander. Genetic and environmental influences on the secondary sex ratio in mice. *Jour. Genet.* 53:200-214. 1955.

6. KALMUS, H., J. D. METRAKOS, and M. SILVERBERG. Sex ratio of offspring from irradiated male mice. *Science* 116:274-275. 1952.

7. KING, H. D. Studies on inbreeding. III. The effects of inbreeding, with selection, on the sex ratio of the albino rat. *Jour. Exp. Zool.* 27:1-35. 1918.

8. MACDOWELL, E. C. and E. M. LORD. Data on the primary sex ratio in the mouse. *Anat. Rec.* 31:143-148. 1925.

9. ————————. The relative viability of male and female mouse embryos. *Am. Jour. Anat.* 37:127-140. 1926.

10. PARKES, A. S. Studies on the sex ratio and related phenomena.—Foetal retrogression in mice. *Proc. Roy. Soc.*, B, 95:551-558. 1924.

11. TRASLER, D. G. and J. D. METRAKOS. Sex ratio of offspring from irradiated male mice. *Rec. Genet. Soc. Am.* Number 22:107. 1953.

12. WEIR, J. A. Association of blood-pH with sex ratio in mice. *Jour. Hered.* 44:133-138. 1953.

13. ————————. Influence of the male on sex ratio of offspring in high and low blood-pH lines of mice. *Genetics* 38:700-701. 1953. (Abstract)

14. ———————— and R. D. CLARK. Production of high and low blood-pH lines of mice by selection with inbreeding. *Jour. Hered.* 46:125-132. 1955.

15. ————————, R. H. COOPER and R. D. CLARK. The nature of genetic resistance to infection in mice. *Science* 117:328-330. 1953.

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HUMAN HEREDITY

HUMAN genetics is a field in which the research worker must still go for much of his data to medical clinics, but it is at last adopting the methodology and the techniques of theoretical genetics and of statistics. The publication of Neel and Schull's book* is a conscious effort by these authors to help in establishing human genetics as a science in its own right. To do this within the confines of a relatively small book is something of a challenge, and the authors have wisely chosen to stress the methodology of the new science rather than to offer a comprehensive review of the facts. It is a clearly written and very readable account of the working problems of the geneticist who chooses the human species as his material. For the serious student of biology or of medicine it sets forth the questions about human heredity which are being asked by geneticists and it gives a few definite answers, some tentative answers, and more often the many reasons why there are no answers yet. For those actively working in the field it points out the difficulties and the cautions that must be taken, and it offers working methods that the authors consider desirable and practicable. The last four chapters in the book, the one on epidemiology, the two on counseling and medicolegal problems (which are the author's specialties), and the final one on eugenics, are well done and deserve careful reading by medical students, doctors, public health workers, and anyone else concerned with the private or public problems affected by human heredity, now or in the future. The authors bring to these problems the constructive but very cautious approach required by our present limited state of knowledge.

The collaboration of two very competent geneticists, one with a medical background and the other with theoretical and statistical preparation, and both now working in the Heredity Clinic of the Institute of Human Biology of the University of Michigan, has many advantages. One of them is that the book is up-to-date in spite of the very wide range of topics that had to be covered. The integration is not so complete as to hide all the seams, but this is not important.

In commenting on the book I find it convenient to treat the 20 chapters as if they were in three sections. The first five chapters form a preliminary section. They introduce the advantages and disadvantages of man as genetic material, describe the physical basis of heredity, explain the genetic diversity of man, deal with nature and nurture relations, and present the elements of probability theory. The complexities of human genetics are explicitly stressed and properly prepare the reader for the later chapters. Chapter 2 on "The Physical Basis of Heredity in Man," is very brief but seems to me out of place in this book. Most readers will skip it but should pause to look at the reproduction of Dr. T. C. Hsu's excellent photograph of human chromosomes.

The middle group of chapters, 6 to 16, deal with: (6) dominant genes in man, (7) recessive genes in man, (8) genes neither dominant nor recessive, (9) the more complex genetic situations involving quantitative inheritance

*_Human Heredity_. James V. Neel and William J. Schull. University of Chicago Press, 1954. 361 pp. $6.00.

including that of intelligence, (10) linkage, in which the modified (3 × 3 table) sib-pair method of Penrose is given, (11) some simple methods of estimating mutation rates (with concrete examples), and the relation between radiation dose and mutation rate, (12) physiological genetics (the biochemical mutants of man), (13) the estimation of gene frequencies by the maximum likelihood method and the chi-square test of goodness of fit with genetic hypotheses, (14) the analysis of family data, (15) population genetics as a method of studying anthropology by examining the blood group gene frequencies of the various races, and (16) twins.

Sex-linked genes have no separate chapter but are treated in chapters 6 and 7. The organization of these chapters is original, but it fails to assign even a section head to the Hardy-Weinberg Law, which it introduced under the heading "The ratio of heterozygotes to homozygotes" in chapter 7 on recessive genes. This section is followed by one on consanguinity which I consider inadequate for a book on human heredity. For instance, the coefficient of relationship between two individuals is dispensed with in a few sentences. Also inadequate from my point of view is the treatment of genetic (random) drift in the chapter on population genetics. After mentioning Birdsell's study of Australian tribes and Laughlin's study of Eskimos, the authors state that "At the present time we have no convincing examples of the operation of genetic drift in man . . .", and conclude (p. 248) that one must resist the temptation to use genetic drift to explain inexplicable differences in two sets of data. However, it should be recorded that one year after the book went to press, Neel and Schull regarded the three-generation data on a small community of Dunkers (Glass, B. H. Advances in Genetics. 6:130-135. 1954) as the best example to date of random drift of gene frequencies in man.

Chapter 13 on "The Estimation of Genetic Parameters and Tests of Genetic Hypotheses" and Chapter 14 on "The Analysis of Family Data" will mean little to readers who must skip the mathematical procedures. This should not deter anyone who reads this book to learn what it is that students of human genetics are working on and what they are finding out. However, any student interested in working in this field or even in understanding it thoroughly, should be able to master the kinds of statistical methods given in these chapters.

The final section of four chapters covering epidemiology, counseling, medicolegal aspects, and eugenics, was referred to earlier.

A good feature of the book is the abundance of illustrative examples drawn from recent data, a considerable proportion of which are from the authors' own research and experience in the Heredity Clinic at the University of Michigan. Examples from animal genetics are drawn on for the sake of clear presentation, but analogies to Drosophila have been reduced to a minimum. This conforms with Neel's philosophy as outlined in his presidential address ("the obvious trap of regarding man as an overgrown fruit fly," *Am. Jour. Human Genet.* 7:1-14, 1955).

Generalities in this book are stated cautiously and coupled with the possibility of alternative interpretations upon further research.

In dealing with man and his environment, the authors after giving several illustrations of the environmental modification of the expression of the genotype, say: "It would be well at an early stage of this book to dispel a common misconception . . . that what is inherited is fixed and immutable, and nothing can be done about it. . . . Carried to an extreme, it leads to a philosophy of biological determinism" (p. 22). So it is natural that assertions and beliefs of the C. D. Darlington variety ("The Facts of Life") are not to be found in this book.

The account of eugenic problems is objective, balanced, and clear. The principle of voluntary sterilization is accepted with sound reasons. However, attention is called to the distinction between the "genetic argument" and the "sociological argument." I think most human geneticists will agree with the authors' concluding remark: "The day may yet come when man has sufficient accumulated knowledge . . . that a comprehensive program of self-directed evolution may be undertaken. But for the present the effort which would be expended on a eugenics program might better go into efforts to explore the many gaps in our present fragmentary information."

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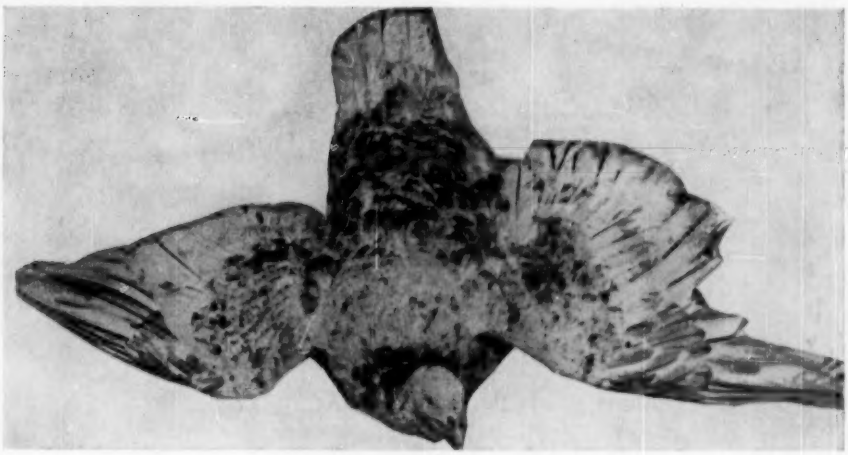
INHERITANCE OF SPOTTING IN THE PLUMAGE OF TURKEYS

V. S. ASMUNDSON*



NEBRASKAN SPOTTED HEN

Figure 14

This turkey shows the typical distribution of black pigment. The plumage on the neck, back and part of the wings shows more pigment than on other parts of the bird.

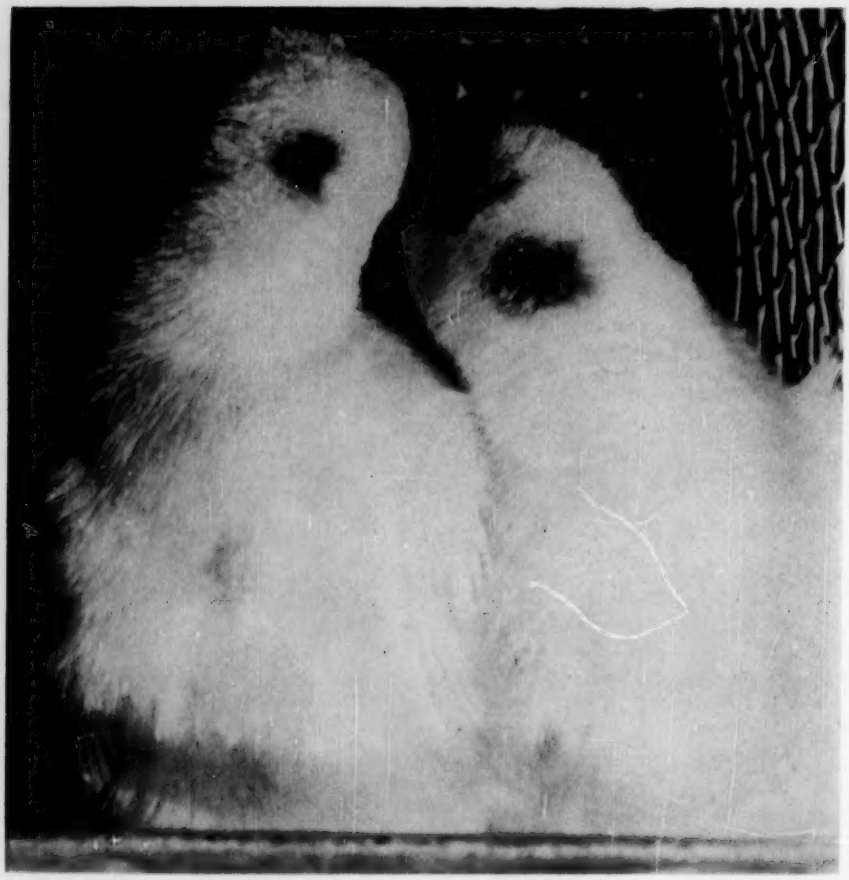
DAY old poults of the Nebraskan variety have white down with a brown head spot or spots (Figure 15). The first plumage is white, the second or juvenile plumage[5, 7] has black pigment scattered through it and the amount of pigment is apparently increased in the adult plumage although none of the feathers are completely black. The amount of pigmentation in plumage on different parts of the bird varies, as it does typically on many color varieties of this species,[9] there being more pigment in the plumage on the neck, back and part of the wings than on other parts of the bird (Figure 14).

In order to investigate the genetics of this mutation, 50 eggs were obtained from a flock of Nebraskan turkeys late in May 1953. Eleven young hatched and nine of these were reared to maturity and used for mating.

Results

Matings of Nebraskan birds produced only spotted progeny, while matings of Nebraskan males with Bronze and with Brown produced only Bronze pattern progeny (Table I). The F_2 progeny of crossbreds from Nebraksan male mated to bronze females comprised 114 non-spotted (Bronze) and 33 spotted (Nebraskan), a good fit to the expected numbers of 110.25 to 36.75 on the basis of a 3:1 ratio, assuming that spotting is a simple autosomal recessive. The numbers obtained from the backcross of F_1 on Nebraskan do not fit expectation as well as the F_2 data but the deviations from expectation are not statisti-

*Department of Poultry Husbandry, University of California, Davis. We are indebted to Mr. A. C. Tuley, Rocklin, California for a donation of Nebraskan eggs.



DAY-OLD NEBRASKAN POULTS

Figure 15

One-day old turkey poults of the Nebraskan variety showing white down except for the brown head spots.

cally significant (P = .08 and .12 that equally great deviations due to chance would be expected).

The hatch of fertile eggs from Nebraskan hens was consistently less than from Bronze or F_1 hens (Figure 16). While there was higher early mortality of embryos in eggs from the Nebraskan hens the greatest difference was in the percentage of dead in shell. These dead embryos were all examined and no lethals were found. If relatively more spotted than nonspotted embryos died, death must have occurred before the down patterns could be identified. The only indication of lower viability of embryos homozygous for spotting is therefore the slight deficiency of spotted progeny from backcrosses on Nebraskan. On the basis of the evidence so far available, it does not seem likely that the gene for spotting,[3] like the one for ex-

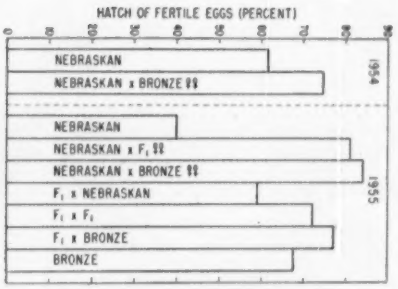


Figure 16

The diagram above illustrates the hatch of fertile eggs from Nebraskan, Bronze and the crosses between them.

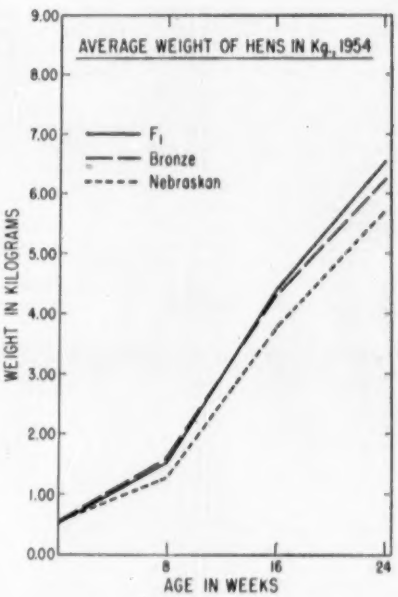


Figure 17

The average weights of Nebraskan, Bronze and F_1 hens are illustrated graphically.

tension of black in the fowl,[3] reduces viability.

Since it is known that inbreeding reduces hatchability,[6] it seems likely that the strain of Nebraskans used for this cross was inbred. This is indicated by the improvement in hatch of fertile eggs when crossed with Bronze or Brown (Figure 16) and by the growth of the F_1 progeny, which was comparable to that of the Broad Breasted Bronze strain used for the cross (Figure 17).

Discussion

The plumage color of the Nebraskan variety of turkeys is determined by an autosomal gene, which may be designated *sp*, recessive to non-spotting or solid color such as found in the Bronze. The *sp* gene appears to be the only one determining the differences between the plumage color of the Nebraskan and the Bronze varieties. The latter may be considered the wild type for this species. The (Bronze) heterozygote from the cross appears to be noticeably browner on the head at day old than the homozygous Bronze although it is not certain that the difference is sufficiently consistent to accurately segregate the two.

The gene for spotting appears to suppress or delay the deposition of black pigment in the plumage of the young bird; the only colored spots found on the newly hatched young Nebraskan are brown, while in the adult brown pigment is not visible. This apparent action of the gene in the homozygote contrasts with that of the b^t gene which, in the homozygote (Black-winged bronze) appears to suppress

TABLE I. Classification of progeny

Matings		Progeny					
		Nonspotted (Bronze)			Spotted		
Sire	Dam	Male	Female	Sex not known	Male	Female	Sex not known
Nebraskan	Nebraskan				19	26	2
Nebraskan	Bronze	58	47				
F_1 (Nebr. × Bronze)	Bronze	11	12	9			
F_1 (Nebr. × Bronze)	Nebraskan	5	2	1		2	1
F_1 (Nebr. × Bronze)	F_1 (Nebr. × Bronze)	45	66	3	15	18	
Nebraskan	F_1	10	5		5	6	
Nebraskan	Nebraskan				16	6	
Nebraskan	Brown	12	11				

brown pigment in the young bird since the down and juvenile plumage do not show brown pigment and the toe nails are white while the amount of brown in the adult plumage appears to be similar to that of the Bronze.[1, 2] The spotting gene restricts pigmentation in a different way from any so far found in turkeys.

A large number of genes for spotting are known in mammals. In birds Jaap[4] has reported on spotting in ducks which show little resemblance to spotting in the Nebraskan variety. Splashed, an autosomal recessive to solid color, in the domestic fowl[8] resembles the spotting in turkeys although pigmentation appears to be more restricted in adult splashed birds than in spotted turkeys.

Summary

The Nebraskan turkey typically has white down with a brown head spot or spots, white first plumage with black pigment spots in later plumages. Nebraskan male mated to Bronze hens produced bronze progeny: in the F_2 progeny the ratio, 114 nonspotted (bronze) to 33 spotted, agreed well with expectation on the assumption that the Nebraskan variety is homozygous for a spotting gene *sp* which is an autosomal recessive to a gene for nonspotting. Down and plumage color of the progeny of backcrosses on the parent varieties agreed with expectation.

The low hatch of fertile eggs found in the strain of Nebraskan tested was apparently not caused by lethal action of the spotting gene.

Literature Cited

1. Asmundson, V. S. Inherited non-barring of the flight feathers in turkeys. *Jour. Hered.* 30:352-358. 1939.

2. ————. A triple-allele series and plumage color in turkeys. *Genetics* 30:305-322. 1945.

3. Hutt, F. B. Lethal action of the gene for extension of black pigment in the fowl. *Genetics* 36:213-234. 1951.

4. Jaap, R. G. Inheritance of white spotting in ducks. *Poultry Sci.* 12:233-241. 1933.

5. Leopold, A. S. The molts of young wild and domestic turkeys. *Condor* 45:133-145. 1943.

6. Marsden, S. J. and C. W. Knox. The Breeding of Turkeys. U.S.D.A. Yearbook of Agriculture. 1350-1366. 1937.

7. Moultrie, F., C. D. Mueller and L. F. Payne. Molting and growth of individual feathers in turkeys exposed to 10 or 24 hours of daily light. *Poultry Sci.* 34:383-388.

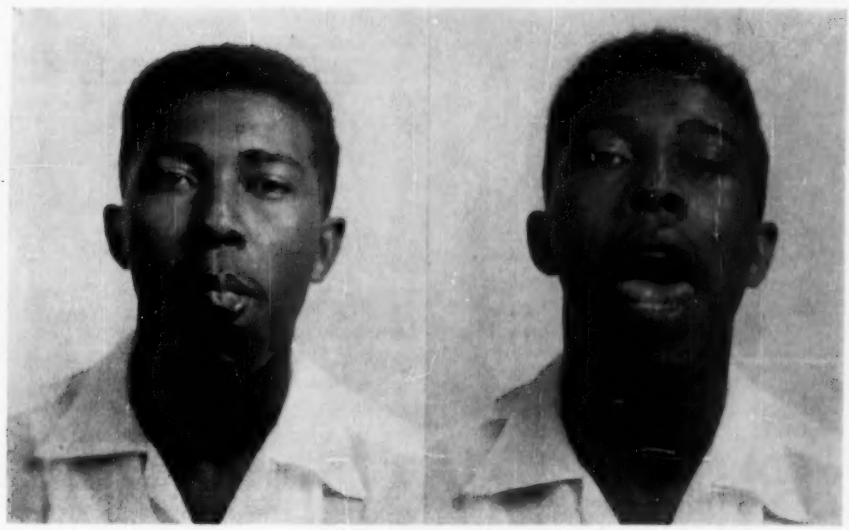
8. Quinn, J. P. A color mutation in the Rhode Island Red fowl. *Jour. Genet.* 29:75-83. 1934.

9. Robertson, W. R., B. B. Bohren and D. C. Warren. The inheritance of plumage color in the turkey. *Jour. Hered.* 34:246-256. 1943.

TONGUE-FOLDING AND TONGUE-ROLLING

In An American Negro Population Sample

J. Warren Lee*



ROLLING AND FOLDING

Figure 18

Demonstrations of the rolling and folding characteristics are shown above. These lingual abilities are used as the criterion for rolling and folding in this study.

THE tongue is a mobile organ, capable of many types of movement. One of these, as reported by Sturtevant[4] is the ability to roll up the lateral edges of the tongue. He suggested that this lingual movement is inherited as a dominant character. Another kind of lingual movement was described by Hsu[1] as a folding of the distal end of the tongue back onto the main body of the organ. The latter movement is performed without employing the use of the teeth. Hsu[1] indicated that upfolding of the lingual appendage may be inherited as a pair of recessive genes. Whitney[5] studied a sample of the white population and corroborated Hsu's[1] findings. Lui and Hsu[2] investigated a sample of the Chinese population to see if rolling and up-folding were inherited independently of each other. They suggested that there is an interaction between the two types of movements. The purpose of this paper is to report the results of a study on the frequencies of rolling and folding in a sample of the American Negro population.

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A survey was conducted at Southern University on a sample of the student population. The abilities to roll and fold the tongue were tested simultaneously on 1,890 students. The subjects examined included 919 students during the summer session of 1954 and 971 freshmen of the 1954-55 term. The symbols *AA* and *Aa* represent the genotypes for rolling, *aa* for non-rolling. The symbols *BB* and *Bb* represent the genotypes for non-folding, and *bb* for folding.

Demonstrations of the rolling and folding lingual characteristics are shown in Figure 1*A* and *B*. Folding the distal end of the tongue back on the main organ (Figure 1*B*) will be used as the criterion for folding during this investigation.

Table I shows that 17.26 percent of the females tested and 10.27 percent of the males in this survey can roll and fold. The table further indicates that 70.79 percent of the males can roll, but not fold, and of the females only 65.25 percent have this ability. Table I shows 2.44 percent of the females and 2.10 percent of the males can fold but not roll. Statistical analyses of these data in Table I indicate no significant differences in the frequencies of rolling and folding with regard to the sexes as indicated by Table I.

Table II shows a comparison of the proportion of tongue-rolling and folding between a sample of the Chinese population and a sample of the Negro population. Hsu[1] indicated that 58.25 percent of his sample could roll but not fold, while the present data show that 68.25 percent of a sample of the Negro population can roll but not fold. In addition, Lui and Hsu[2] indicated that only 3.26 percent could roll and fold, while in the Negro population 13.70 percent are in this class. Lui and Hsu[2] failed to find any individuals that could fold but not roll, while the present data show that 2.27 percent of those tested have this ability.

As shown in Table II, it appears that there are slight differences between the two populations. These differences may be due to the rigid criterion used by Lui and Hsu[2] in determining folding. If this is true, then the differences may not be significant. The data, however, fail to demonstrate the classical Mendelian ratio 9:3:3:1, for two randomly assorted genes. Such a ratio would be expected only in a population in which the allele frequencies of *A* and *a* and those of *B* and *b* were 0.5, that is, where *A* and *a* and *B* and *b* are of equal frequencies.

Table III shows that there is 18 percent of the recessive class of non-rolling individuals (341 out of a total 1,890), and 16 percent (320 out of a total 1,890) with the recessive

TABLE III. Test for independence of two tongue movement characters

	Roll	Not roll	
Fold	259 (247.5)*	43 (54.5)*	302
Not fold	1290 (1301.5)*	298 (286.5)*	1598
χ^2 = about 3.52 P = about .07	1549	341	1890

*The figures in parentheses are those expected on the null-hypothesis that the frequencies of roll to not-roll are the same in the fold and not-fold groups.

TABLE I. Frequency of two tongue movements in relation to sex

	Can roll and fold		Can roll but not fold		Can neither roll nor fold		Can fold but not roll		Total	
Sex	Actual No.	Percent	Actual No.	Percent	Actual No.	Percent	Actual No.	Percent	Actual No.	Percent
Female	149	17.26	563	65.24	130	15.06	21	2.44	863	100
Male	110	10.72	727	70.79	168	16.39	22	2.10	1027	100
Total	259	13.99	1290	68.02	298	15.78	43	2.27	1890	100

TABLE II. Comparison of the proportion of tongue rolling and folding in two different populations

		Can roll but not fold		Can roll and fold		Can neither fold nor roll		Can fold but not roll		Total	
	Genotype	*A — B*		*A — bb*		*aa — B*		*aa — bb*			
Author	Population	Actual No.	Percent	Actual No.	Percent	Actual No.	Percent	Actual No.	Percent	Actual No.	Percent
Present Data	Negro	1290	68.25	259	13.70	298	15.78	43	2.27	1890	100
Lui and Hsu	Chinese	615	58.96	34	3.26	394	37.78	0		1043	100

trait for folding. According to the Hardy-Weinberg law the allele frequencies P_a and P_b are to be obtained by $P_a = \sqrt{.18}$ and $\sqrt{.16}$, thus giving 42 percent respectively. The gene frequencies are the basis of the distribution of the population.

Lui and Hsu[2] failed to find any individuals who could fold but not roll, and therefore assumed an interaction of genes *A* and *b*. Table III shows that 43 such individuals were found in the population sample in this study. The present data (Table III) are compatible with independence in the expression of the two pairs of characters, although there are some deficiencies in the fold-not-roll class.

Although the P value is greater than five percent, it is rather low and the relatively high chi-square 3.52 is mostly due to a deficiency in the fold-not-roll class. The data suggest a possibility of a high degree of independence between the rolling and folding characters.

Summary

A gene frequency study on the occurrence of rolling and folding in a sample of the American Negro population was made. The data suggest a possibility of a high degree of independence between the rolling and folding characters.

Literature Cited

1. Hsu, T. C. Tongue unfolding. *Jour. Hered.* 39:187-188. 1948.

2. Lui, T. T. and T. C. Hsu. Tongue-folding and tongue-rolling. *Jour. Hered.* 40:19-21. 1949.

3. Snyder, L. H. The principles of gene distribution in human populations. *Yale Jour. of Biol. and Med.* 19:819-833. 1947.

4. Sturtevant, A. H. Tongue-rolling. *Proc. Nat. Acad. Sci.* 26:100-102. 1940.

5. Whitney, D. D. Tongue tip overfolding. *Jour. Hered.* 40:18-19. 1949.

◆

SYMPOSIUM ON EFFECTS OF RADIATION AND OTHER DELETERIOUS AGENTS ON EMBRYONIC DEVELOPMENT

This symposium* was sponsored by the Biology Division of the Oak Ridge National Laboratory and was held at Oak Ridge, Tennessee, April 20 and 21, 1953. An idea of the comprehensive nature of this symposium can be obtained by glancing through the table of contents:

Introduction—Alexander Hollaender; James G. Wilson, Differentiation and the reaction of rat embryos to radiation; Roberts Rugh, The effect of ionizing radiations on amphibian development; James N. Dent, and Ernest L. Hunt, Radiotracer techniques in embryological research; Liane Brauch Russell and W. L. Russell, An analysis of the changing radiation response of the developing mouse embryo; Samuel P. Hicks, The effects of ionizing radiation, certain hormones, and radiomimetic drugs on the developing nervous system; Dietrich Bodenstein, Effects of radiomimetic substances on embryonic development, with special reference to nitrogen mustards; Josef Warkany, Disturbance of embryonic development by maternal vitamin deficiencies; F. Clark Fraser, H. Kalter, B. E. Walker and T. D. Fainstat, The experimental production of cleft palate with cortisone and other hormones; Walter Landauer, On the chemical production of developmental abnormalities and of phenocopies in chicken embryos; B. H. Willier, Phases in embryonic development; James N. Yamazaki, Stanley W. Wright and Phyllis M. Wright, A study of the outcome of pregnancy in women exposed to the atomic blast in Nagasaki; Paul Weiss, Summarizing remarks.

Although this is a work on embryonic development there are a number of features in it that are of considerable interest to geneticists. One of the most interesting things is the finding that there are definite critical periods for the application of these mutagenic agents whether they be X-rays, nitrogen mustards, or other teratogenic agents. Rather large doses were required to produce the abnormalities that were studied. Wilson found that below 200 roentgens delivered on the eighth day of the life of a rat embryo mortality did not increase in a graded fashion as the dosage increased but suddenly became complete at 200 r. Radiation at the eighth day in the life of the embryos produced no malformations. The major effect at this stage of the rather undifferentiated embryo was one of mortality which became complete at 200 r, also the body weight of embryos was slightly reduced. At later stages such as the 9th, 10th and 11th days the amount of radiation required to kill 100 percent of the embryos increased to 600 r. However, at these later stages there were many abnormalities produced.

The rat embryo at eight days is rather undifferentiated and differentiation proceeds

*Proceedings published in the *Journal of Cellular and Comparative Physiology*, Vol. 43, Supplement 1, May 1954.

rapidly from the ninth to the eleventh days. As differentiation increases the susceptibility for the production of abnormalities by radiation apparently increases also. Wilson stated that the capacity of the rat embryo to react to irradiation undergoes a qualitative change between the eighth and ninth days of gestation, and a quantitative change from the ninth through the 11th day, with decreasing susceptibility to radiation as age progressed.

In the mouse embryo the Russells found roughly three stages in the development of the embryo. The pre-implantation stage lasting from one-half to four and one-half days, the period of major organogenesis lasting from about five days to about 13 days and the period of the fetus lasting from about 13 days to 16½ days. In the first stage or the pre-implantation stage 200 r caused no increase over the control in the amount of abnormalities. The only effect at this stage was prenatal death, which was very marked. The second stage contained a high percentage of abnormalities at the same dose of 200 r. Neonatal death, that is still-birth and death within 24 hours, occurred over a very narrow range from about seven to 11 days. Radiation before and after these times produced no increase over the control.

The results of the Russells and also of Wilson showed that the response to radiation differed markedly during the progress of the life of the embryo from fertilization to birth. These differences in sensitivity have a parallel in differences in sensitivity in developing gametes in both plants and animals. The work of Mary Lou Alexander, Clayton and Stone have shown marked differences in the developing germ cells of *Drosophila* in their susceptibility to X-irradiation. Likewise we have found in corn that there is a big difference in the susceptibility in different stages of the meiotic cycle, those approaching the mature pollen being much more susceptible to surviving mutations than those near the period of meiosis. Also, the mature pollen grains show a marked drop in susceptibility to chronic gamma radiation the same as mature *Drosophila* sperm show to a marked decrease in susceptibility to X-rays.

In the rat embryo it was also found that hypoxia protects markedly against radiation induced abnormalities. This is similar to a protection of hypoxia against chromosome breaks induced in various organisms.

One of the effects of nitrogen mustard as studied by Dietrich Bodenstein is that mitotic activity is inhibited and that a number of giant cells results.

There is a comprehensive paper by Walter Landauer on the chemical production of developmental abnormalities and of phenocopies in chicken embryos. It is possible with chemical methods to produce syndromes of malformations which bear close resemblance to certain mutant conditions and which for this reason have been referred to as phenocopies. Some of the chemicals used in his treatments were sulfamilimide, eserine, insulin, pilocarpine, boric acid, etc. He stated that "It has long been known as a result of studies of many kinds of organisms that there are definite well-defined stages of embryonic development during which certain environmental agencies will change the fate of particular parts." This has been referred to in the case of mouse and rat embryos. Also, a quotation from Landauer stated "A good deal of evidence is available for the conclusion that the genetic constitution of an animal is of great importance for the outcome of experiments with teratogenic substances." I think this applies not only to teratogenic substances but to X-rays. The Russells in their work found considerable difference between different strains of mice in their susceptibility to X-radiation. Also, it is known that in certain species of plants, wheat and corn for example, there are different genic differences for susceptibility to X-radiation.

In the study of pregnancies in women exposed to the atomic blast in Nagasaki it was shown that among 30 mothers within 2000 meters of the hypo-center, with one or more major signs of radiation damage the percent of fetal deaths, neonatal and infants deaths were higher than for mothers without major radiation damage, or mothers from the "control" area (4000-5000 meters from the hypo-center). Also, the four cases of mentally retarded children occurred in the group of 30 mothers.

The mean height and head circumference of children born to mothers with major signs was significantly smaller than in those children born to mothers in the control group.

These studies demonstrated that radiation of pregnant mothers had a serious effect on the course of pregnancies and on the infants born. The radiation level was high, since they were within 2000 meters from the explosion. Even the control group, 4000-5000 meters from the hypo-center, would have received considerable radiation, yet they were assumed to be normal enough to be used as controls, showing no measurable effect of the radiation received.

The excerpts given from this volume are by no means comprehensive. It seemed a most worth-while symposium. Anyone interested in embryonic development will find much of interest in it. Geneticists will find certain similarities between induced effects on embryonic development, and effects on chromosomal disturbances and hereditary effects.

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CORDOVAN, A NEW ALLELE OF BLACK AND BROWN COLOR IN THE MOUSE

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THE genes producing black and brown coat color in the house mouse appear to be exceedingly stable. Despite the widespread use of C57 black and brown strains, to our knowledge, only one other possible allele, "light," described by MacDowell,[1] has been reported previously. The purpose of this paper is to report the spontaneous occurrence of a new mutation affecting coat color, which appears to be an allele of black (*B*) and brown (*b*); it is an autosomal gene, recessive to black and dominant over brown.

In March, 1953, in an F_1 litter from a cross of a C57BL/10 male by a DBA/1 female, one female appeared which was distinctly deep brown in color in contrast to the normal black color of her littermates. Since the color is the rich brown of cordovan leather, the mutation was called "cordovan" (symbol b^c). The C57BL male which sired this cordovan female was in the 13th generation of pure stock maintained by brother-sister matings in our colony. The original C57 stock was obtained from the Jackson Laboratory in 1946. The same male sired six other F_1 litters (five by the same female), in none of which cordovan appeared.

Genetics of Cordovan Coat Color

The original cordovan female was bred to her C57BL father, and produced an all black litter of seven females and one male. Four of these females were mated with their black brother; cordovan individuals appeared in all the resulting litters, proving that the gene was real and present.

The cordovan female was then mated with her black son, producing four black, three cordovan, one blue, and one dilute brown offspring. Since the cordovan female was an F_1 individual, her genotype appeared to be *Ddb^{c}b*. The appearance of the blue and dilute brown offspring indicated that the black son carried the dilution gene (*d*). The dilute brown offspring at first led us to believe that the genotype of the black son was *DdBb*. However, the fact that, when bred to his mother, to his four black sisters, and to DBA/1 females, he never sired a brown offspring (*Ddbb*) made this interpretation unlikely. Furthermore, the litters of all four black sisters sired by this male contained cordovan mice, and it was improbable that all four females carried the cordovan gene (expectation $\frac{1}{2}$). Therefore, we were led to the hypothesis that the dilute brown coat color could result from the genotype *ddb^{c}b*, and that the genotype of the black son was *DdBb^c*. This postulate, that the dilute brown phenotype can be dilute cordovan, was later demonstrated (Tables IV-VI).

Six of the seven black daughters (one died after her first litter) were subsequently mated with pure DBA/1 males, as was the cordovan female. The distribution of coat color among their progeny is presented in Table I. The ratios of expected phenotypes shown in parentheses are those expected on the following assumptions:

1) Cordovan color is produced in the presence of intense (*D*) and cordovan (b^c), and in the absence of black (*B*).

2) In the presence of *B*, gene b^c has no effect on phenotype, since black is completely dominant. Mice of genotype *DDBb^c* or *DdBb^c* are black; *ddBb^c* mice are blue (maltese).

3) The dilution gene causes dilution

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of cordovan (either heterozygous or homozygous) to a color indistinguishable from dilute brown (DBA); *ddb^{c}b* and *ddb^{c}b^e* mice show dilute brown coat color.

It is interesting to note the distribution of genotypes among the eight black offspring of the cordovan female when mated with her C57/10 father. If the father transmitted only normal C57 color genes (*D* and *B*), the following four genotypes should be equally represented in the litter: *DDbb^e*, *DDBb*, *DdBb^e*, *DdBb*. This expectation was closely approximated; the actual ratio was 2 *DDBb^e* : 2 *DDBb* : 1 *DdBb^e* : 3 *DdBb*.

From the data in Table I, it is apparent that the black offspring of the cordovan female may carry the dilution gene (*d*) and *either* brown (*b*) *or* the new allele *b^e*. In crosses with DBA, any black female which has brown offspring cannot produce cordovan progeny (♀♀ 3, 4, 5, 7). Conversely, in any cross, a black mouse which has cordovan progeny cannot have any of brown color (♀♀ 1, 2, 6, ♂8). Thus it appears that cordovan is a true allele of black and brown, which has replaced *B* in the cordovan mutation.

In Table I, it will be noted that in some cases, (as in the progeny of the cordovan female), the color distribution was surprisingly close to the expected values even in small numbers. In other cases, the expected ratios were not achieved. However, if the offspring of females of the same genotype are pooled, the distribution of coat color comes close to the ratios expected on the basis of a monohybrid cross.

TABLE I. Distribution of coat color among progeny of original cordovan female and of her seven black daughters and black son. Expected ratios are given in parentheses

Parent	× DBA (*ddbb*)	× *DdBb^e* (♂ #8)	Probable genotype
Cord. ♀	9 cord.: 7 brown: 15 DBA (1: 1: 2)	4 blk.: 3 cord.: 1 blue: 1 DBA (3: 3: 1: 1)	*Ddb^{c}b*
Blk. ♀ #1	10 blk.: 5 cord. (1: 1)	6 blk.: 2 cord. (3: 1)	*DDBb^e*
Blk. ♀ #2	(died)	6 blk.: 2 cord. (3: 1)	*DDBb* (?)
Blk. ♀ #3	4 blk.: 3 brown 5 blue: 2 DBA (1: 1: 1: 1)	.....	*DdBb*
Blk. ♀ #4	14 blk.: 8 brown (1: 1)	.....	*DDBb*
Blk. ♀ #5	3 blk.: 2 brown: 2 blue: 2 DBA (1: 1: 1: 1)	.....	*DdBb*
Blk. ♀ #6	7 blk.: 12 cord. (1: 1)	12 blk.: 4 cord. (3: 1)	*DDBb^e*
Blk. ♀ #7	4 blk.: 3 brown 1 blue: 4 DBA (1: 1: 1: 1)	4 blk.: 2 cord.: 3 blue: 1 DBA (1: 1: 1: 1)	*DdBb*
Blk. ♂ #8	16 blk.: 11 cord. 9 blue: 11 DBA (1: 1: 1: 1)	.....	*DdBb^e*
♀♀ 1 + 6	17 blk.: 17 cord. (1: 1)	18 blk.: 6 cord. (3: 1)	*DDBb^e*
♀♀ 3,5,7	11 blk.: 8 brown: 8 blue: 8 DBA (1: 1: 1: 1)	.....	*DdBb*

Crosses of Cordovan Mice

Several matings of doubly heterozygous cordovan males and females (*Ddb^{c}b* × *Ddb^{c}b*) were made in order to observe whether the expected 9: 3: 4 (F_2) ratio of phenotypes would appear among their progeny. Most of these cordovan mice were derived from crosses of cordovan with pure DBA; any cordovans resulting from such a cross were of known heterozygous genotype. In addition, there were a number of matings of cordovan males and females of other genotypes. The results of these cordovan crosses are summarized in Table II.

Cordovan mice of all four possible genotypes were mated with pure DBA mice. The resulting progenies are shown in Table III.

All of these crosses involving cordovan mice resulted in the ratios of phenotypes expected on the assumption that cordovan is an allele of brown. It should be noted that there has been no reappearance of black among the progeny of any cordovan mouse crossed with cordovan, DBA or blue.

TABLE II. Distribution of coat colors among progenies of different types of matings between cordovan mice. Expected numbers are given in parentheses

Type of mating	No. of matings	Expected ratio	Coat color of progeny Cord.	Brown	DBA	Total
Ddb^{c}b × *Ddb^{c}b*	6	9:3:4	90 (90)	29 (30)	40 (40)	159
Ddb^{c}b × *DDb^{c}b*	1	3: 1	27 (27)	8 (9)	....	35
Ddb^{c}b × *Ddb^{c}b^e*	2	3: 1	31 (30)	....	10 (10)	41
Ddb^{c}b^e × *Ddb^{c}b^e*	2	3: 1	19 (19½)	....	7 (6½)	26
DDb^{c}b^e × *Ddb^{c}b^e*	5	all	46 (46)	....	....	46
DDb^{c}b^e × *Dfb^{c}f*	2	all	36 (36)	....	....	36

Interaction of Dilution and Cordovan

In interpreting the breeding results presented in Table I, we made the assumption that cordovan is diluted to the same extent as is brown by the presence of the dilution gene, so that mice showing the typical "dilute brown" coat color may actually be dilute cordovan (*ddb^{c}b* or *ddb^{c}b^c*). In order to test this postulate, suspected dilute cordovan mice were mated with brown, to introduce the intensity gene necessary for the appearance of cordovan coat in their progeny. The available brown mice were F_2 and backcross individuals (from C57/10 × DBA/1), most of which were heterozygous.

Eight dilute brown mice were tested, which from their parentage (*Ddb^{c}b^c* × *ddbb*) must, under our hypothesis, carry the cordovan gene. All eight had cordovan offspring in the expected proportion. Fourteen animals which from their parentage (*Ddb^{c}b* × *ddbb*, *Ddb^{c}b* × *Ddb^{c}b*) could either carry or lack the cordovan gene were mated with brown. Of these, nine transmitted cordovan to their progeny, while five did not. Eight mice showing dilute brown coat color proved to be homozygous dilute cordovan (*ddb^{c}b^c*).

In a total of 26 mice, the dilute brown coat color was found to be associated with dilution of cordovan. The results of the test matings of these mice with brown are presented in Tables IV, V, and VI.

It is evident that mice showing "dilute brown" coat color may carry and transmit the cordovan gene. These breeding results also support the hypothesis that cordovan is an allele of black and brown, since the ratios obtained are those resulting from a monohybrid cross.

In our experience the phenotypes dilute brown and dilute cordovan are indistinguishable. One mating of a dilute cordovan male and female (*ddb^{c}b* × *ddb^{c}b*) produced a litter of eight, all showing the typical dilute brown coat color. Breeding tests with brown mice indicated that two were dilute brown (*ddbb*), three were heterozygous dilute cordovan (*ddb^{c}b*) and three were homozygous dilute cordovan (*ddb^{c}b^c*).

In these and other crosses, there was no evidence of sex linkage. The coat colors were equally distributed among male and female progeny (which were therefore pooled), and reciprocal crosses gave similar results.

Blue (Maltese) Mice Carrying Cordovan

A further point to be tested is the assumption that the complete dominance of black over cordovan provides the possibility that blue mice may carry the cordovan gene in combination with black. Blue mice of genotype *ddBb^c* should be present in the progenies of several of our matings, as in the cross of a dilute cordovan male by an F_1 female (*ddb^{c}b* × *DdBb*). Eight blue mice which could carry the cordovan gene were mated with brown or F_1 mice in order to observe whether cordovan coat color appeared among their offspring.

TABLE III. Distribution of coat colors among progeny of cordovan mice mated with DBA mice. Expected numbers are given in parentheses

Type of mating Sire	Dam	No. of matings	Expected ratio	Coat color of progeny Cord.	Brown	DBA	Total
Ddb^{c}b	*ddbb*	4	1:1:2	9	6	19	34
ddbb	*Ddb^{c}b*	7		43	41	73	157
			Total	52	47	92	191
				(48)	(48)	(95)	
DDb^{c}b	*ddbb*	3	1:1	22	21	....	43
ddbb	*DDb^{c}b*	2		15	16	....	31
			Total	37	37	....	74
				(37)	(37)		
Ddb^{c}b^c	*ddbb*	6	1:1	31	....	29	60
ddbb	*Ddb^{c}b^c*	6		37	....	45	82
			Total	68	....	74	142
				(71)		(71)	
DDb^{c}b^c	*ddbb*	8	all	59	....	....	59
ddbb	*DDb^{c}b^c*	4		32	....	....	32
			Total	91	....	....	91
				(91)			

TABLE IV. Distribution of coat colors among progeny of heterozygous "dilute cordovan" mice (*ddb^{c}b*) when mated with heterozygous brown mice (*Ddbb*)

Mating #	Coat color of progeny Cord.	Brown	DBA	Total	Parents of dil. cordovan
24	2	1	1	8	*Ddb^{c}b^c* × *ddbb*
49, 51	11	11	23	45	*ddb^{c}b* × *ddb^{c}b*
48, 55	8	7	19	34	*Ddb^{c}b* × *ddbb*
58, 60	10	12	19	41	*Ddb^{c}b^c* × *ddb^{c}b*
86, 112	7	6	19	32	*ddb^{c}b* × *DdBb*
91, 95	10	9	22	41	*Ddb^{c}b* × *Ddb^{c}b*
Total	48	48	105	201	
Expected	50¼	50¼	100½		$\chi^2 = 0.403$

TABLE V. Distribution of coat colors among progeny of homozygous "dilute cordovan" mice (*ddb^{c}b^c*) mated with heterozygous brown mice (*Ddbb*)

Mating #	Mating Sire	Dam	Color of progeny Cord.	DBA	Total	Parents of dil. cordovan
50, 63	*Ddbb*	*ddb^{c}b^c*	15	11	26	*ddb^{c}b* × *ddb^{c}b*
56, 57	*ddb^{c}b^c*	*Ddbb*	11	15	26	*ddb^{c}b* × *ddb^{c}b*
59	*Ddbb*	*ddb^{c}b^c*	14	12	26	*Ddb^{c}b^c* × *ddb^{c}b*
62, 125	*ddb^{c}b^c*	*Ddbb*	7	10	17	*Ddb^{c}b^c* × *ddb^{c}b*
94	*Ddbb*	*ddb^{c}b^c*	12	14	26	*Ddb^{c}b* × *Ddb^{c}b*
		Total	59	62	121	
		Expected	60½	60½		$\chi^2 = 0.074$

TABLE VI. Distribution of coat colors among progeny of dilute cordovan mice mated with homozygous brown mice (*DDbb*). Expected numbers are given in parentheses

Mating #	Color of progeny Cord.	Brown	Total	Genotype of dil. cord.	Parents of dil. cordovan
61, 101	37 (37)	....	37	*ddb^{c}b^c*	*Ddb^{c}b^c* × *ddb^{c}b*
106, 107	12 (10)	8 (10)	20	*ddb^{c}b*	*ddb^{c}b* × *ddb^{c}b*
130	7 (6½)	6 (6½)	13	*ddb^{c}b*	*Ddb^{c}b^c* × *ddbb*

Of the eight blue mice tested, four produced cordovan progeny, while four did not. The results of these matings are shown in Table VII.

Although the numbers of progeny in these matings are not adequate to warrant quantitative conclusions, the trends are clear. Qualitatively, the expected phenotypes appeared in all matings, with no exceptions. No brown offspring appeared among the 118 produced by blue mice of genotype *ddBb*^{c}, and conversely, no cordovans appeared among the 84 progeny of blue mice which produced brown offspring. It is clear that phenotypically blue mice may carry the cordovan gene in combination with the black gene, again indicating the dominance of black over cordovan.

Cordovan appears to be a rare mutation. Because of the dominance of black, the highest probability of detection of the mutant is in mice bearing only one black gene (genotype *DDBb* or *DdBb*). In our colony, we have bred more than 1000 F_1 mice from C57/10 by DBA/1 and DBA/2, 1000 F_2 animals (of which three-eighths bear these genotypes), 2000 mice from the backcross to C57 (one-half *DDBb* and *DdBb*), and 2000 from the backcross to DBA (one-fourth *DdBb*). In addition, an equal number of mice in the same crosses derived from C57/6 have been bred. In this large population, we have observed the cordovan mutation only once.

We are now in the process of developing homozygous cordovan mice in order to preserve the mutant type.

TABLE VII. Distribution of coat colors among progeny of blue (dilute black) mice which could carry the cordovan gene. Expected numbers are given in parentheses

Mating Sire	Mating Dam	Coat color of progeny Blk.	Cord.	Brown	Blue	DBA	Total	Genotype of blue parent
Blue	*DdBb*	17 (16)	6 (6)	....	11 (16)	7 (6)	43	*ddBb*^{c}
Ddbb	Blue	17	9	....	6	15	47	*ddBb*^{c}
Blue	*Ddbb*	4	7	....	4	5	20	*ddBb*^{c}
	Total	21 (17)	16 (17)	....	10 (17)	20 (17)	67	
Blue	*DDbb*	5 (4)	3 (4)	....	....	....	8	*ddBb*^{c}
Blue	*Ddbb*	3	....	2	3	1	9	*ddBb*
Blue	Blue	15	....	15	13	16	59	*ddBb*
	Total	18 (17)	....	17 (17)	16 (17)	17 (17)	68	
	DDbb	10 (8)	....	6 (8)	....	....	16	*ddBb*

Summary

A new mutant affecting coat color in the house mouse has been described. Cordovan (symbol b^{c}) appears to be an allele of black and brown. It is an autosomal gene, recessive to black and dominant over brown. The new coat color is a rich deep brown. In combination with the dilution gene, cordovan is diluted to the same degree as is brown, resulting in a coat color indistinguishable from "dilute brown." In the presence of the black gene, cordovan causes no alteration of the blue (dilute black) phenotype. All ratios obtained were those expected on the basis of monohybrid crosses, indicating that cordovan is an allele at the black locus, rather than a modifying gene.

Literature Cited

1. MacDowell, E. C. "Light," a new mouse color. *Jour. Hered.* 41:35-36. 1950.

A CYTOLOGICAL STUDY OF THE INDUCED OCTOPLOID

Of an *Agropyron-Hordeum* Hybrid

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THIS study is chiefly concerned with the meiotic chromosome behavior of a fertile, colchicine-induced octoploid derived from a sterile, tetraploid hybrid between *Agropyron trachycaulum* (Link) Malte and *Hordeum jubatum* L. A significant feature of this investigation lies in the fact that the origin of the hybrid is known and the cytology of its progenitors is reasonably well understood.[1]

Materials and Methods

A potted plant of the sterile tetraploid hybrid was broken up and the clones placed in a flat of wet sand and permitted to form vigorous root systems. These were removed, washed and the roots cut back to a length of approximately three inches. The roots of each clone were inserted into a large test tube containing a 0.1 percent aqueous solution of colchicine for 24 hours after which they were washed and potted in fertile soil. Thirteen clones were treated in this manner. One of these subsequently produced two octoploid culms bearing 35 caryopses. These germinated immediately upon planting and the resulting F_2 generation furnished the material for this study.

Anthers were fixed in 3:1 absolute alcohol-acetic acid or in Newcomers[5] solution. The latter proved far superior. Smears were made in aceto-carmine. Observations and photographs were made from temporary mounts.

Meiotic Chromosome Behavior

Diakinesis and Metaphase I. One-hundred thirty-seven cells were interpreted (Figure 19*A* and *B*). A summary of the various chromosome associations is shown in Table I. Bivalents were present in all cells and averaged 24.6 per cell. The majority (80.4 percent) were ring bivalents. Univalents occurred in 50.3 percent of the cells and averaged 1.3 per cell. Their frequency ranged from zero to six per cell. Quadrivalents occurred in 52.5 percent of the cells and averaged 0.7 per cell. Their frequency ranged from zero to three per cell. Trivalents were found in 13.8 percent of the cells. Only one cell contained more than one trivalent. Although 20 different types of chromosome association were observed 80 cells (58 percent) fell into three types.

Anaphase I. Thirty-three cells at anaphase I were examined (Table II). Twenty-two of the cells (66.7 percent), showed at least one laggard. The number per cell varied from zero to four with an average of 1.4 per cell. No bridge-fragments were found in this stage. With the exception of the laggards the divisions were regular although in several instances precocious division of dyads was observed (Figure 20*B*).

Anaphase II. Forty-two cells at anaphase II were examined (Table II) Laggards were found in this stage in a considerably greater number than in anaphase I; 90.5 percent of the cells contained at least one laggard, with an average of 3.1 per cell. (Figure 19*A*).

Tetrads. One-hundred and twenty-seven tetrads were examined and 92.9 percent contained at least one micronucleus. Micronuclei averaged 5.8 per tetrad.

Pollen grains. Of the 10,254 pollen grains examined 44 percent were abortive. Among the good pollen grains a variation in size was noted.

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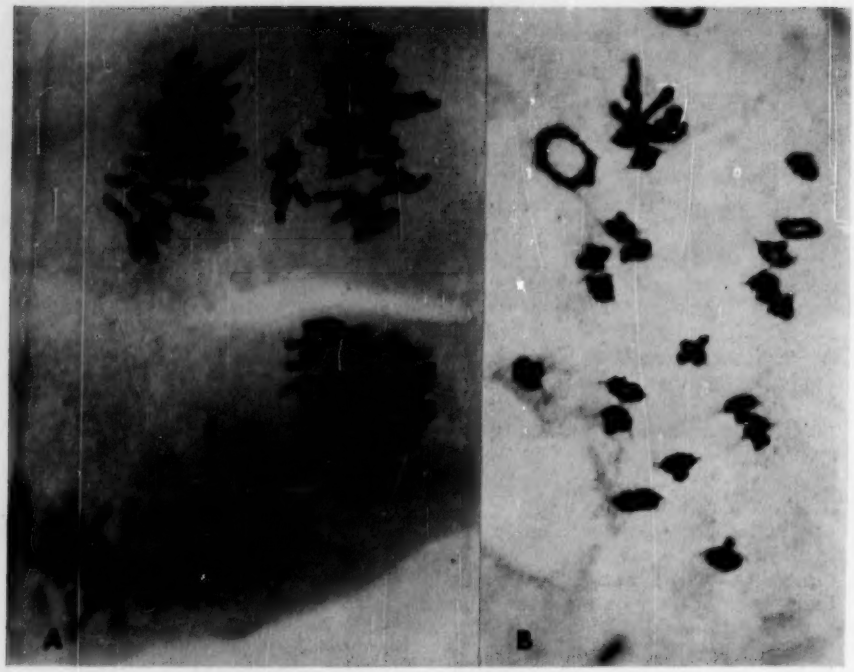


ANAPHASE II AND METAPHASE I

Figure 19

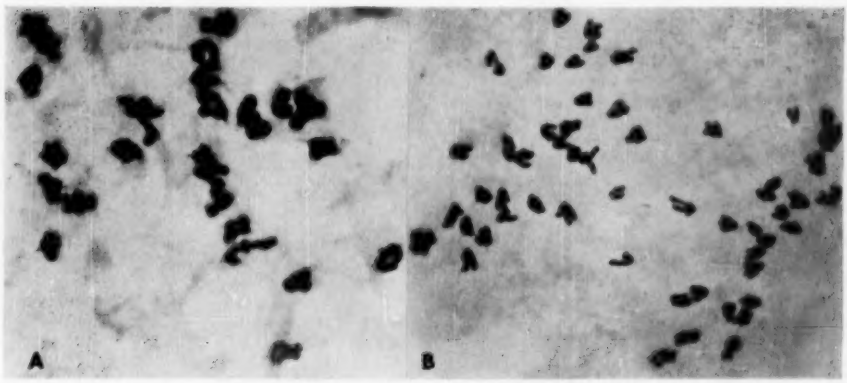
Meiotic stages in the colchicine-induced octoploid. *A*—Anaphase II, lagging chromosomes at equatorial plate, 912×. *B*—Metaphase I, 1_{IV}, 25_{II}, and 2_{I}, 1194×.

Discussion

Cytological studies of the sterile tetraploid hybrid[1] indicate that *Agropyron trachycaulum* and *Hordeum jubatum* probably have one genome in common. The formulae *AABB* and *AACC* were suggested for the parents and *AABC* for the tetraploid hybrid. On this basis the genomic formula for the colchicine-induced octoploid may be represented as *AAAABBCC*.

In the light of the above assumptions certain speculations can be made concerning chromosome associations that can be expected to occur in the octoploid. Fourteen chromosomes should have only one homologue, and thus constantly show 14 bivalents at metaphase I. Seven chromosomes should have three homologues, thus making possible a variety of associations ranging from 14 bivalents to seven quadrivalents.

The postulated genomic formula was largely substantiated by the chromosome associations found at diakinesis and metaphase I, as can be seen from Table I. As would be expected, bivalents were the most common association and 35 cells were found in which the chromosome associations consisted entirely of 28 bivalents. The chromosome association at the other end of the range, 14 bivalents plus seven quadrivalents, was not found in any of the cells examined. This could not be considered as unusual however, for even in autopolyploids the maximum possible number of multivalents is rarely found in a single cell[4]. If two or three of the four homologues pair completely the possibility of quadrivalent formation is eliminated and the remaining chromosomes form a bivalent or univalent. Of the 28 chromosomes in this plant presumably capable of being associated in multivalents it is obvious that the probability of this occurring in a single cell is slight. The maximum number of chromosomes found associated in multivalents in any one cell was 13 (three trivalents and one quadrivalent). This particular association was found in only one cell. However, three cells were observed that contained 12 chromosomes associated in multivalents, all as three quadrivalents.



METAPHASE I AND ANAPHASE I

Figure 20

A—Metaphase I, 28_{II}, 1374×. *B*—Anaphase I, precocious dyad division, 1244×.

The occurrence of univalents, which averaged 1.5 per cell, is difficult to explain when it is considered that each chromosome should have at least one identical homologue. However, the occurrence of univalents in amphidiploids has been reported by several investigators.[3, 8, 9] Sears[9] has made the observation that all 56 chromosome wheat amphidiploids reported have univalents at meiosis and reduced fertility and that the probable cause of the univalent formation is the large number of chromosomes which act as mechanical barriers to complete pairing. However, as Pope and Love[6] pointed out, the high chromosome number of some plants, such as *Agropyron elongatum* and *Spartina townsendii* do not interrupt the regularity of their meiotic divisions to any significant extent.

Sears[8] stated that multivalent formation can be expected to cause the formation of some univalents. However, as he further points out, in some amphidiploids produced by him univalent occurrence was too frequent to be explained by multivalent occurrence alone, and that these cases could best be explained as the result of physiological unbalance. A similar explanation was favored by Pope and Love.[6] In amphidiploids produced by these investigators, pairing failure was attributed to disharmonious gene interaction and genetically controlled inhibition of pairing.

In this plant, the occurrence of multivalents in 75 percent of the cells is adequate to explain the frequency of univalents and probably offers the best explanation. Dobzhansky[2] discussed univalent formation caused by multivalents and stated that where chromosome duplication occurs in excess of the diploid number pairing competition arises with the resultant formation of uni-, bi- and multivalents in inconsistent proportions from cell to cell.

The occurrence of laggards at anaphase I was to be expected on the basis of univalent occurrence at metaphase I, since unpaired chromosomes generally lag at anaphase. Snyder[10] in his studies on *Elymus* reported an increase of laggards at anaphase I over the observed number of univalents at metaphase I. He explained this by noting that following disjunction fragments appeared at anaphase and behaved as laggards. In our material no fragments were observed in this stage, and the close correlation between the average number of univalents at metaphase I (1.5) and the average number of laggards at anaphase I (1.4) suggests that the origin of the laggards is solely from the univalents.

In several instances at anaphase I the precocious division of a dyad was observed similar to that reported in a *Lilium* hybrid by Richardson.[7] Such divisions were explained as probably having resulted from a bivalent that failed to reach the equator at metaphase and thus disjunction of the bivalent did not occur until the other chromosomes were well into anaphase. One of the dyads from this late disjunction may separate at the centromere and the two sister chromatids move to opposite poles. It is possible that a similar process is involved in the precocious dyad divisions observed in this plant. (Figure 20*B*).

Anaphase of the second division also showed laggards, but in a frequency too high to be explained as a result of first division laggards (Figure 19*A*). Assuming all the laggards at anaphase I divided equationally this could account for a laggard frequency at anaphase II only as high as that present at anaphase I. Any increase over this number must come from another source. Snyder[10] obtained similar data in *Elymus* hybrids. He explained this increase on the basis of additional fragmenta-

tion. This explanation is again not applicable to this plant since no fragments were found in the second division either. In this plant a possible explanation for increase of laggards at anaphase II is by the precocious division of dyads observed in anaphase I. The sister chromatids which separate at anaphase I behave as univalents, and thus laggards, through the second division. An additional source of laggards in the second division was the occasional occurrence of dyads that had not separated in the division and were found occurring as laggards.

Bridge fragment configurations were found at anaphase II but only in three cells. The low frequency of these configurations and their complete absence in the first division suggests that the cause is something other than a cross-over in a heterozygous inversion. It is possible, as shown by Richardson,[7] to have a cross-over in a heterozygous inversion that does not form the typical chromosome bridge until the second division. For this to occur it necessitates heterozygosity for a paracentric inversion in which two cross-overs take place, one in the inverted segment and one between the inverted segment and the centromere. If this occurs the fragment appears at anaphase I, but the bridge does not appear until anaphase II. It is unlikely that the crossovers should only occur in this precise manner or not at all, and that the typical bridge-fragment configuration should not occur at least as often at anaphase I. In addition, in no instance was a fragment found at anaphase I. A more likely explanation is that spontaneous breakage and reunion of sister chromatids occurred, such as that described by Walters[13] in an interspecific *Bromus* hybrid, thus causing a bridge to form at anaphase II.

The correlation between the number of micronuclei per tetrad and the number of laggards at anaphase II was good. The average number of laggards per cell at anaphase II was 3.1, and since a tetrad consists of two of these cells the average number of micronuclei per tetrad should be approximately 6.2 if the laggards are the source of the micronuclei. The observed frequency of 5.8 micronuclei per tetrad indicates that anaphase II laggards are probably the sole source of the micronuclei. It was noted that the size of the micronuclei was variable. The larger micronuclei probably had their origin from dyads and the smaller from single chromosomes since both occurred as laggards in the second division.

The high frequency of aborted pollen grains is undoubtedly caused to a large extent by the laggards at anaphase I, and the resulting micronuclei. It appears however, that other sterility factors are also present. In high polyploids the loss of some chromatin material is not as serious as in a diploid, because, as Stebbins[12] has pointed out, a high degree of chromosome duplication precludes the necessity of absolute meiotic regularity and makes possible the formation of viable gametes even with the loss of chromosomes or chromosome segments. Based on the average number of micronuclei per tetrad (5.8) the average number per single pollen grain would be approximately 1.5. If this represents the average number of chromosomes excluded and thus lost from the nucleus in a pollen grain, a 44 percent decrease in fertility of an octoploid is scarcely explainable on this basis alone.

A contributing factor to the reduced fertility may be unequal distribution of chromosomes in multivalent associations. However, the results of Sears'[8] investigations tend to show that there is no direct relationship between multivalent frequency and degree of fertility.

The presence of unfavorable genetic factors may be another source of reduced fertility. To what extent each of the above are involved in reducing the fertility of this plant is difficult to determine and it is possible that all are involved in varying degrees. As Stebbins[11] has pointed out the causes of sterility in polyploids are still obscure.

Despite the reduced fertility each octoploid plant in the present study is easily capable of producing several hundred viable caryopses which germinate immediately and produce vigorous plants. "Seed" set was approximately 30 percent under greenhouse conditions. The presence of caryopses is the only prominent morphological characteristic distinguishing

TABLE I. Chromosome association at diakinesis and metaphase I

	I	II	III	IV	Number of cells
	0	28	0	0	15
	0	26	0	1	27
	2	25	0	1	18
	4	26	0	0	11
	3	25	1	0	7
	2	27	0	0	6
	1	26	1	0	5
	2	23	0	2	4
	0	24	0	2	4
	4	22	0	2	4
	1	24	1	1	3
	4	24	0	1	3
	2	21	0	3	2
	0	22	0	3	2
	3	21	1	1	1
	3	21	1	2	1
	5	19	3	1	1
	5	20	1	2	1
	6	23	0	1	1
	6	25	0	0	1
Average: per cell	1.3	25.6	0.15	0.7	137 Total no. cells

TABLE II. Frequency of lagging chromosomes at anaphase I and II

	No. cells	Percent with laggards	Average number laggards
Anaphase I	33	66.7	1.4
Anaphase II	42	90.5	3.1

the octoploid from the sterile tetraploid progenitor.

Summary

The meiotic chromosome behavior is reported for a moderately fertile, colchicine-induced octoploid derived from a completely sterile, tetraploid hybrid between *Agropyron trachycaulum* and *Hordeum jubatum*. Although 20 different types of chromosome associations were observed 58 percent fell into three types: 28 II; 26 II, 1 IV; 25 II, 1 IV, 2 I. Bivalents were present in all cells and averaged 25.6 per cell. The genomic formula *AAAABBCC* is suggested. Pollen abortion was 44 percent and seed set approximately 30 percent under greenhouse conditions. Evidence is described indicating that lagging chromosomes are chiefly responsible for the reduced fertility. Despite the reduction in fertility each plant is easily capable of producing several hundred viable seeds.

Literature Cited

1. Boyle, W. S. and A. H. Holmgren. A cytogenetic study of natural and controlled hybrids between *Agropyron trachycaulum* and *Hordeum jubatum*. *Genetics* 40:539:545. 1955.

2. Dobzhansky, T. Genetics and the Origin of Species. 3rd ed., rev. Columbia University Press. New York. 364 pp. 1951.

3. Love, R. M. and C. A. Suneson. Cytogenetics of certain *Triticum-Agropyron* hybrids and their fertile derivatives. *Amer. Jour. Bot.* 32:451-456. 1945.

4. Myers, W. M. and H. D. Hill. Studies of chromosomal association and behavior and occurrence of aneuploidy in autotetraploid grass species, orchard grass, tall oat grass, and crested wheatgrass. *Bot. Gaz.* 102:236-255. 1940.

5. Newcomer, E. H. A new cytological and histological fixing fluid. *Science* 118:161. 1953.

6. Pope, W. K. and R. M. Love. Comparative cytology of colchicine-induced amphidiploids of interspecific hybrids: *Agropyron trichophorum* × *Triticum durum*, *T. timopheevi* and *T. macha*. *Hilgardia* 21:411-426. 1952.

7. Richardson, M. M. Structural hybridity in *Lilium martagon album* × *L. hansonii*. *Jour. Genet.* 32:411-450. 1936.

8. Sears, E. R. Chromosome pairing and fertility in hybrids and amphidiploids in the Triticinae. *Missouri Agri. Expt. Sta. Res. Bul.* 337. 20 pp. 1941.

9. ————. The amphidiploids *Aegilops cylindrica* × *Triticum durum* and *A. ventricosa* × *T. durum* and their hybrids with *T. aestivum*. *Jour. Agric. Res.* 68:135-144. 1944.

10. Snyder, L. A. Cytology of inter-strain hybrids and the probable origin of variability in *Elymus glaucus*. *Amer. Jour. Bot.* 38:195-202. 1951.

11. Stebbins, G. L., Jr. The evolutionary significance of natural and artificial polyploids in the family Gramineae. *Hereditas*, Suppl. Vol. 461-485. 1949.

12. ————. Variation and Evolution in Plants. Columbia University Press. New York. 643 pp. 1950.

13. Walters, M. S. Spontaneous breakage and reunion of meiotic chromosomes in the hybrid *Bromus trinii* × *B. maritimus*. *Genetics* 35:11-37. 1950.

THE INHERITANCE OF RED AND BLUE HIND TIBIAE

In the Lesser Migratory Grasshopper, *Melanoplus mexicanus mexicanus* (Saussure)

ROBERT L. KING AND ELEANOR H. SLIFER*

DESPITE the very extensive and detailed studies of the chromosomes of the Acrididae, little is known of the genetics of this family. White[14] lists only two papers which deal with acridian genetics. One is by Sansome and La Cour[11] on color patterns in *Chorthippus parallelus* and the other by Creighton and Robertson[3] on color patterns in the closely-related, and perhaps identical, species, *Chorthippus longicornis*. In addition to these a short abstract by King[9] states that the melanic form of *Melanoplus differentialis* results from the presence of an autosomal, recessive gene in the homozygous condition. Carothers[2] reported, also in an abstract, that she had studied (1) the markings on the tegmina, (2) the width and extent of the wing band and (3) the color of the hind tibiae in the offspring of crosses between *Trimerotropis citrina* and *T. maritima*. No details are given. Slifer,[13] in a footnote, comments that in *Melanoplus differentialis* it is possible to obtain, by selection, stocks which produce eggs with no diapause and others with eggs with a very long diapause. Multiple factors seem to be involved.

In his original description of the lesser migratory grasshopper Saussure[12] states that the metathoracic tibiae are red but that individuals with blue hind tibiae also occur. This condition has been recorded frequently by later observers. Hebard[6] in a study of 1,178 males and 467 females from Montana reports that "Of these, 267 males and 155 females have the caudal tibiae glaucous, a small number glaucous proximad and pink distad or wholly straw color, the remainder pink." This same investigator[5] examined 409 adults from South Dakota and concludes that "In the more humid sections of the state the great majority have the caudal tibiae pink, very few having these members pale glaucaus. In the more arid sections, the reverse is true." A small sample of the same species from Alberta[7] consisted of 15 individuals with pink hind tibiae, 11 with glaucous and one with tibiae which were purplish proximad and glaucous distad. Another group from Kansas[8] contained 100 with red hind tibiae and 32 with glaucous. Mexican material, Hebard[4] notes, contains a higher proportion of specimens with glaucous hind tibiae than does that from the United States. Blatchley[1] describes the lesser migratory grasshopper as having hind tibiae which are "normally pale red, but in about 8 percent of the specimens they are either red at tip and otherwise colored, or dull yellow, or glaucous, the males having them more variable in hue than the females."

Description

In 1953 an opportunity to study the factors responsible for these striking differences in the color of the metathoracic tibiae of *Melanoplus mexicanus mexicanus* occurred. A laboratory stock of this species had been established during the preceding year from wild specimens caught near Iowa City, Iowa. Most of the adults which developed from the eggs laid by the wild individuals had red hind tibiae but a few had blue. The blue-legged grasshoppers were removed to a separate cage as soon as they had molted for the last time and the red-legged adults were, likewise, all caged together.

One-hundred and forty-six adults, all with blue hind tibiae, were reared from the eggs laid by the first generation selected for blue. Of these, 79 were females and 67 were males. One-hundred and forty-four adults were obtained from the red stock. Of these, 135 were

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red (60 females and 75 males) and nine were blue (two females and seven males). The results indicated that blue hind tibiae, in this species, might be due to a single, autosomal, recessive gene present in the homozygous condition and that red could be due to the presence of the corresponding dominant allele.

Before attempting to cross the stocks both were carried through another generation. The blue stock, as before, gave all blue (78 females and 74 males) while the red stock gave all reds (16 females and 20 males). Since the number of reds was small, and in view of the results obtained in the preceding generation, it was expected that some of the red individuals were homozygous and others heterozygous for the dominant gene.

A number of females were isolated from each stock as soon as they had molted for the last time and these were used to make up single pair matings with males having tibiae of the opposite color. Fifty-six adult offspring were obtained from five matings of this type and the results are summarized in Table I.

On the assumption that blue is due to a recessive, autosomal gene in the homozygous condition and that red is due to the presence of its dominant allele all of the F_1 red individuals should be heterozygous. Accordingly, the red offspring from pair one were caged together as soon as they became adult and the red offspring from pair two and pair three were treated in the same manner. The results of these three matings are shown in Table II. A number of the eggs laid by these three pairs were sent to Dr. Donald H. Bucklin, at the University of Wisconsin, who wished to establish a stock of this species. He reported that of 88 adults which developed from these eggs 69 had red hind tibiae and 19 had blue. These results are included in Table II.

When all of the results for the three matings are combined the deviation from a 3:1 ratio for red:blue is less than two and the F_2 results support the assumption made earlier that the inheritance of red and blue metathoracic tibiae in *Melanoplus m. mexicanus* is controlled by a single pair of autosomal genes.

Up to this point nothing has been said about variations in the red and blue colors found in this stock. Hebard[4, 5, 6, 7] has described the tibiae as red, pink, glaucous, glaucous proximad and pink distad, purplish proximad and pink distad and as wholly straw color. In the present study no difficulty was encountered in assigning an individual to either the red or the blue category although in each group color variations were found. The metathoracic tibiae of the red grasshoppers varied from geranium red to rose red when compared with Ridgway's color standards.[10] The blue tibiae varied over a much wider range from glaucous blue through pale glaucous green to a very pale sulphur yellow. To discover whether or not the color of the blue tibiae changed with age 14 females and seven males of the blue stock were examined immediately after the final molt and the tibial color recorded. At this time the color ranged from glaucous blue to a very pale sulphur yellow. Each individual was kept in a separate container and examined again at the end of two weeks. Almost no change had occurred in the colors except that all of those first described as very pale yellow were now pale glaucous green, that is, a faint blue had been added to the yellow. Nine of these individuals were kept until they were four weeks old but no further color changes were detected. No difference in variability within the two sexes was apparent. Several of the blue grasshoppers were killed with KCN and then dried to see whether or not the procedures to which museum specimens are usually subjected would affect the color of the tibiae but no changes were detected.

Hebard[6, 7] recorded the occurrence in a few specimens of tibiae which were of different colors at the proximal and distal ends. Several of this type were noticed in the first generation of the present stock but, unfortunately, their importance was not realized at that time and they were not preserved. No others appeared in the following generations.

Although red and blue hind tibiae are evidently controlled by a single pair of genes in this species the possibility is not excluded that environmental conditions may have some influence on the exact color which develops. However, all of the specimens in the present experiments were reared under similar conditions and each of the colors listed could be found in individuals from a single cage. It seems probable that modifying genes may be responsible for the color variations found in the two classes. Nevertheless, and in view of Hebard's statement quoted earlier concerning the predominance of red in humid areas and of blue in more arid regions, it would be interesting to raise a large number of individuals

TABLE I. F_1 offspring from five crosses between parents with red and with blue metathoracic tibiae

Pair	Parents	Red offspring	Blue offspring
1	Blue ♀ × red ♂	1 ♀♀ + 2 ♂♂	1 ♀ + 1 ♂
2	Blue ♀ × red ♂	7 ♀♀ + 6 ♂♂	1 ♀
3	Red ♀ × blue ♂	14 ♀♀ + 8 ♂♂	none
4	Red ♀ × blue ♂	6 ♀♀ + 4 ♂♂	none
5	Red ♀ × blue ♂	4 ♀♀ + 1 ♂	none

TABLE II. F_2 offspring from three matings of red F_1 individuals

F_2 from pair	Red offspring	Blue offspring
1	25 ♀♀ + 23 ♂♂	9 ♀♀ + 10 ♂♂
2	27 ♀♀ + 34 ♂♂	15 ♀♀ + 11 ♂♂
3	19 ♀♀ + 23 ♂♂	4 ♀♀ + 5 ♂♂
Totals	71 ♀♀ + 80 ♂♂	28 ♀♀ + 28 ♂♂
	151 red	56 blue
Additional F_2 data from Dr. Bucklin	69 red	19 blue
Combined totals	220 red	75 blue

of each of the two stocks under a variety of conditions to see whether a definite environmental effect can be superimposed on the genetic effect.

Summary

Evidence has been obtained that red metathoracic tibiae in *Melanoplus mexicanus mexicanus* (Saussure) are dependent on the presence of a dominant, autosomal gene and that blue tibiae are produced when its recessive allele is present in the homozygous condition.

Red tibiae were found to vary from geranium red to rose red in this species and blue tibiae from glaucous blue to pale sulphur yellow. Since the full color range was found in individuals raised in a single cage it seems probable that modifying genes may be responsible for the variations within the red and the blue stocks.

Literature Cited

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2. Carothers, E. E. Interspecific hybridization in the Acrididae (*Trimerotropis citrina* × *T. maritima*) *Genetics* (abstract). *26*:144. 1941.

3. Creighton, M. and W. R. B. Robertson. Genetic studies on *Chorthippus longicornis*. *Jour. Hered.* 32:339-341. 1941.

4. Hebard, M. Notes on Mexican Melanopli (Orthoptera; Acrididae). *Proc. Acad. Nat. Sci. Phila. 69*:251-275. 1917.

5. ————. The Orthoptera of South Dakota. *Proc. Acad. Nat. Sci. Phila.* 77:33-155. 1925.

6. ————. The Orthoptera of Montana. *Proc. Acad. Nat. Sci. Phila.* 80:211-306. 1928.

7. ————. The Orthoptera of Alberta. *Proc. Acad. Nat. Sci. Phila.* 82:377-403. 1930.

8. ————The Orthoptera of Kansas. *Proc. Acad. Nat. Sci. Phila.* 83:119-227. 1931.

9. King, R. L. Inheritance of melanism in the grasshopper, *Melanoplus differentialis*. *Am. Nat.* (abstract) 67:80. 1933.

10. Ridgway, R. Color Standards and Color Nomenclature. Washington, D. C. 1912.

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10. Ridgway, R. Color Standards and Color Nomenclature. Washington, D. C. 1912.

11. Sansome, F. W. and L. LaCour. The genetics of grasshoppers: *Chorthippus parallelus*. *Jour. Genet.* 30:415-422. 1935.

12. Saussure, H. de. Orthoptera Nova Americana (Diagnoses praeliminares). *Rev. et Mag. de Zool.* 13:156-170. 1861.

13. Slifer, E. H. The effects of xylol and other solvents on diapause in the grasshopper egg; together with a possible explanation for the action of these agents. *Jour. Exp. Zool.* 102:333-356. 1946.

14. White, M. J. D. Cytogenetics of Orthopteroid Insects. Advances in Genetics 4: 267-330. 1951.

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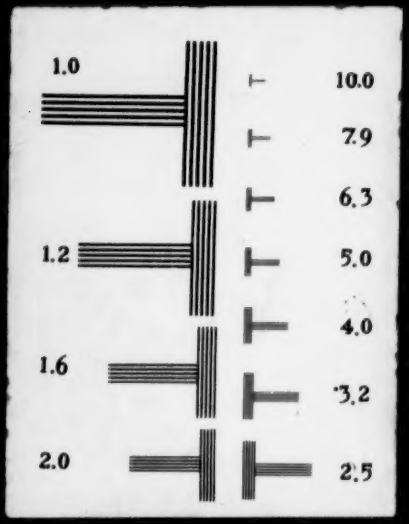

100 MILLIMETERS

INSTRUCTIONS Resolution is expressed in terms of the lines per millimeter recorded by a particular film under specified conditions. Numerals in chart indicate the number of lines per millimeter in adjacent "T-shaped" groupings.

In microfilming, it is necessary to determine the reduction ratio and multiply the number of lines in the chart by this value to find the number of lines recorded by the film. As an aid in determining the reduction ratio, the line above is 100 millimeters in length. Measuring this line in the film image and dividing the length into 100 gives the reduction ratio. Example: the line is 20 mm. long in the film image, and $100/20 = 5$.

Examine "T-shaped" line groupings in the film with microscope, and note the number adjacent to finest lines recorded sharply and distinctly. Multiply this number by the reduction factor to obtain resolving power in lines per millimeter. Example: 7.9 group of lines is clearly recorded while lines in the 10.0 group are not distinctly separated. Reduction ratio is 5, and $7.9 \times 5 = 39.5$ lines per millimeter recorded satisfactorily. $10.0 \times 5 = 50$ lines per millimeter which are not recorded satisfactorily. Under the particular conditions, maximum resolution is between 39.5 and 50 lines per millimeter.

Resolution, as measured on the film, is a test of the entire photographic system, including lens, exposure, processing, and other factors. These rarely utilize maximum resolution of the film. Vibrations during exposure, lack of critical focus, and exposures yielding very dense negatives are to be avoided.